COMMONWEALTH OF VIRGINIA)
)
COUNTY OF FAIRFAX)

AFFIDAVIT OF GUILLERMO URIARTE

I, Guillermo Uriarte, make the following statement under penalty of perjury:

1. I was one of the lawyers retained by Trudy Muñoz Rueda to defend against felony child abuse charges stemming from Noah Whitmer's hospitalization on April 20, 2009. The bulk of my legal practice has been immigration cases; the remainder is criminal defense work. My criminal defense experience has mostly involved significantly less serious charges. Prior to this case, I had tried approximately six felony cases, only ~~two~~ one of which were jury trials. This case was the longest felony jury case I had ever tried. ~~Both of~~ My prior felony jury cases ~~, each~~ lasted less than one day.

2. I know Trudy because I am a distant familial relation of her husband, Hernani Ames. I was first introduced to Trudy and Hernani by my father. We were not particularly close prior to this case.

3. I was first contacted by Hernani on April 21, 2009, which was the day after Noah's hospitalization. That day, Trudy was interrogated at her home by a social worker (Joslyn Waldron) and a detective (Nancy Cottrell). Following the interrogation, Trudy was advised by Cottrell that she would need to go down to the police department to turn herself in for arrest. I advised Trudy that going to the police department voluntarily was in her best interest, and accompanied both her and her husband to the police department.

4. Initially, I believed that this would be a minor case, likely involving a misdemeanor child abuse charge. It was not until I called Greg Holt, the Assistant Commonwealth's Attorney assigned to this case, and asked about a possible plea bargain with no jail time that I learned the charges were more serious. Holt laughed when I inquired about reducing the charges so that Trudy would serve no jail time, noting that the child was in the hospital. Holt never offered any kind of plea bargain in this case, advising me at the preliminary hearing that the only "offer" he would make was for Trudy to plead guilty with no reduction in charges and no agreement as to sentencing.

5. Due to my relative inexperience with the issues in a serious felony case, I subsequently sought the help of James Kearney, a personal injury lawyer with whom I had worked on a prior, non-criminal case. I knew Kearney, though a civil lawyer, had experience cross-examining medical professionals, and thought he would be helpful, since I had no experience working with medical records, experts, or doctors.

6. Kearney first became involved in the case shortly before we filed the second motion to attempt to have Trudy released on bond. Kearney assisted in filing this motion and got the Peruvian embassy to testify that Trudy's passport had been surrendered and would not be re-issued, so she would be unable to leave the country.

7. Kearney was not involved in preparing for trial and did not assist me in finding the experts. Kearney was really brought on only to conduct the cross-examination of the Commonwealth's experts. I advised Kearney which experts we would be using; he prepared them in the week before trial.

8. Initially, I believed I had a strategy for this case. However, once Kearney came on, I routinely deferred to his suggestions, given his age and my estimation of his experience in these matters. Particular examples of this included not calling Eva Valle, Trudy's sister-in-law and assistant at the daycare, to testify, as well as not pursuing more thoroughly my questioning of the police officers who initially interviewed Trudy (both of which are discussed below).

9. This was the first significant case in which I had worked with Greg Holt. At first, Holt seemed open to sharing his files with me, though once I asked him to provide me with a copy of the 911 call, he became very obstructionist and insisted that he did not have a copy of that. Eventually, after Holt's numerous refusals to provide the 911 recording, I told Holt "if you don't have it, you don't have it." Trudy told me that, when she called 911, she explained that Noah had gone limp and stopped breathing while Trudy was feeding him. Trudy also told me that the 911 dispatcher talked her through administering CPR to Noah on the speakerphone, even helping her count off the compressions, and tried to calm her down. I believed that the 911 call contained important exculpatory evidence. Dr. Uscinski also told me that he thought it was important to have that call.

10. Despite repeated requests, Holt ~~refused to provide a copy~~ denied the existence of a recorded interview conducted by two homicide detectives with Trudy on April 20, 2009 until very shortly before trial. Additionally, Holt did not disclose all of the medical records in the case until mid-December 2009, despite trial being set for January 2010. Notably, I received neither the MRI nor CT scan images, which were necessary for me to secure and prepare experts, until December 2009, when the bulk of Noah's medical records were provided. I may have looked through the medical records briefly, but my review of them was not comprehensive. I ultimately relied on Kearney and the experts to tell me what was relevant and what our medical defense should be.

11. Ultimately, I contemplated that this case was most likely to be a "battle of the experts," and so retaining Kearney and medical experts who could speak to the medical issues in the case was very significant.

12. In anticipation of trial, I contacted Drs. Ronald Uscinski, Horace Gardner, Kirk Thibault, and Patrick Barnes to inquire about their willingness to serve as experts in Trudy's case. I found their names simply by looking at expert witnesses called by defense attorneys in other, similar cases. Each of the doctors agreed to testify, and Drs. Uscinski, Gardner, and Thibeault were retained and paid a fee in exchange for their services. I offered to pay Dr. Barnes a fee, but he declined one, and explained that he was willing to work on this case for free. These four experts were the only experts I contacted in connection with this case.

13. I forwarded the medical records sent by the Commonwealth to each of the experts approximately a week after receiving them. This was in late December 2009. I spoke with Drs. Uscinski, Barnes, and Gardner in a conference call approximately one and a half weeks before trial was to commence. Kearney was not involved in this conversation. The experts discussed the medical records; I did not fully understand their discussion, but they agreed that Noah's symptoms were not caused by trauma or abuse.

14. Until the week of trial, I intended to use Dr. Barnes as an expert witness.. I believe that Dr. Barnes had to testify in another case and therefore was not available to testify in our trial.

15. Despite the late arrival of the medical records, I never considered asking for a continuance of the trial date. I had no reason not to ask for one, and, it obviously would have been helpful to give both Kearney and our experts sufficient time to review the evidence, research, prepare and communicate their theory to us. I felt that we were rushed into trial and that our preparation in the weeks after discovery was provided was frantic. I was unsure of what to do at that point, asking for a continuance was not something I considered.

16. I believe that the defense medical experts could have helped us put forward a unified medical defense that was consistent with Noah's symptoms and medical history, if they simply had more time to review the medical records and consult one another.

17. I was very concerned about Joslyn Waldron, the social worker who intended to testify for the Commonwealth that Trudy had acknowledged shaking the baby. Waldron had testified at the preliminary hearing, and I was therefore aware of what she intended to say at trial. In order to question her credibility, I tried to address her once in Spanish when I encountered her in the courthouse, to test her fluency in the language. She simply refused to speak to me. I also noted, and brought up at trial, that her hand-written notes, which were in English, seemed to be scattered, as though she had cherry-picked what to write down.

18. I did not have a plan for how to discredit Waldron at trial, other than questioning her language capabilities and her note-taking of the interview with Trudy.

19. My intent was to call Eva Valle to testify about Waldron's interrogation of Trudy. Ms. Valle witnessed the interrogation of Trudy by Waldron and Cottrell. Ms. Valle could have testified to the pressure and coercive tactics being used during the interrogation. Instead, I deferred to Kearney, who thought we should not call Ms. Valle, because he was the more senior attorney.

20. Although I did interview Renata Ames, Trudy's daughter, who was fourteen at the time, and considered calling her as witness, I never asked her about her interactions with Noah prior to April 20, 2009. I only considered calling Renata because I believed that the jury would find her to be a sympathetic witness. Renata also witnessed the interrogation of Trudy by Waldron and could have described to the jury what happened consistent with Trudy's testimony and would have contradicted Waldron on the key issue of whether Trudy ever said or demonstrated that she shook Noah.

21. I did put Trudy's husband, Hernani Ames, on the stand to testify about the length of time Trudy was interrogated, but I did not think to ask him about what was actually said at the end of the interrogation, when he arrived at the house. I did not realize at the time that Hernani had important information about the fact that what the social worker was characterizing as shaking was actually a very gentle motion that could not possibly have injured Noah. Had I known that Hernani saw Trudy demonstrate how she held and rocked Noah and that he heard Waldron and Cottrell characterize that as "shaking," I would certainly have presented that evidence to contradict Ms. Waldron's testimony.

22. I did not interview Renata Ames about Noah's behavior during the week prior to April 20, 2009. I likewise did not thoroughly review Noah's medical records for other possible explanations for his symptoms. Given the late disclosure of Noah's medical records by the Commonwealth, my review of them was rushed. I simply did not have a lot of time to review them in detail. I primarily relied on our experts to review the medical records and alert me to any important information in them.

23. Since I received Noah's medical records less than a month before trial and sent them to the experts still after that, I was not able to present the experts' coherent medical explanation that connected the facts regarding Noah's fussiness the week before to the facts present in his medical record. Thus, I did not think about the possibility of putting Ms. Valle on the stand to discuss Noah's behavior the week before, nor did I think about interviewing Renata Ames about her interactions with Noah the weeks before.

24. The area of investigation in which I was most interested concerned the homicide detectives. The tape-recorded interview with the detectives from April 20, 2009 lasted for more than two hours. At the end of the tape, Trudy leaves the room, and the detectives can be heard whispering amongst themselves about one of the children. The child was purportedly excited by all the commotion. This child

was apparently the son of another police officer, Mike Byrnes. To me, it seemed as though the detectives were concerned that Byrnes's son might be implicated in injuring Noah Whitmer. Because of this, I believed the detectives elected to focus their efforts on Trudy in order to divert blame from their colleague. As I did not interview any of the homicide detectives before trial, I had hoped to bring out at trial both statements about their initial concerns and the fact that they had called Byrnes that day to discuss the situation with him. My goal was not to imply that Byrnes's son had actually injured Noah. Rather, I wished to imply that the detectives believed this was a possibility, prompting them to focus more intently on Trudy and thereby showing bias.

24. Kearney, however, did not believe questioning the detectives at trial was an important issue, and encouraged me not to call Byrnes to the stand. I never spoke directly with Byrnes or any of the homicide detectives about their substantive testimony prior to trial. Byrnes and the detectives were very upset that I had subpoenaed them for trial.

25. I spoke with a number of parents whose children were in Trudy's care about their experiences with Trudy generally, in order to evaluate them as character witnesses. I did not speak with any of these parents in an effort to determine whether Trudy had talked to any of them about what had happened on April 20, 2009. All of the parents gave glowing recommendations of Trudy's work with their children, with no exceptions. Nevertheless, I called only one character witness. I had no strategic reason for not calling all of them.

25. Additionally, Eva previously told me that she had noticed a bruise on Noah's head sometime during the week prior, and had alerted both Trudy and the child's mother, Erin Whitmer, to this fact. Eva told me that Mrs. Whitmer had said that he must have bumped his head at home. Additionally, Eva told me that Noah was cranky and fussy over the prior week, and that at one point his poop was both copious and green. When Eva advised Mrs. Whitmer of this, the mother explained that they had recently switched the child to solid food, which accounted for his crankiness. Eva and Trudy commented to each other during the week prior to April 20, 2009 that the baby might be teething, because he seemed very upset and unable to be calmed.

26. I understand that signing an affidavit is like testifying in court. I have been given the opportunity to make any corrections before signing this affidavit and have carefully reviewed it.

FURTHER AFFIANT SAYETH NAUGHT.

GUILLERMO URIARTE

Signed and sworn before me this 13TH day of November, 2012.

NOTARY PUBLIC

My commission expires: 12-31-2014

Notary registration no.: 359386

NORMA D. AGUILAR
NOTARY PUBLIC
COMMONWEALTH OF VIRGINIA
MY COMMISSION EXPIRES DEC. 31, 14

EXHIBIT R

STATE OF CALIFORNIA)
)
CITY OF PALO ALTO)

AFFIDAVIT OF PATRICK BARNES, MD

I, Dr. Patrick Barnes, make the following statement under penalty of perjury:

1. I am Patrick David Barnes, MD, an actively practicing pediatric neuroradiologist over the past 35 years. Currently, I am Chief of the Section of Pediatric Neuroradiology and Co-Director of the Pediatric MRI and CT Center at the Lucile Packard Children's Hospital (LPCH) and Stanford University Medical Center (SUMC), Palo Alto, California, and Professor of Radiology, Stanford School of Medicine (2000-2012). I am also Co-founder and Member of the Child Abuse Task Force and SCAN Team for LPCH and SUMC. Previously, I was Chief of the Division of Neuroradiology and Director of MRI at the Children's Hospital, Boston, Massachusetts, and Associate Professor of Radiology, Harvard Medical School (1986-1999). Prior to that I was a pediatric radiologist and Chief of Pediatric Neuroradiology at Oklahoma Children's Memorial Hospital and Associate Professor, University of Oklahoma College of Medicine (1977-1986). I received my Doctrate of Medicine (MD) from the University of Oklahoma College of Medicine 1973, followed by residency training in Diagnostic Radiology, including Pediatric Radiology, at the University of Oklahoma College of Medicine (1973-1976), and then fellowship training in pediatric neuroradiology and cardio- vascular radiology at the Boston Children's Hospital (1976-1977). I am previously licensed to practice medicine in the states of Oklahoma and Massachusetts, and am currently licensed in the state of California. I have been certified by the American Board of Radiology in Diagnostic Radiology (1977) with a certificate of added qualification in Neuroradiology (1995) including continuing maintenance of certification in Neuroradiology (MOC 2008). My curriculum vita and child protection services resume are attached.

2. I was contacted by Guillermo Uriarte in 2009, and asked if I would review the medical records in the Trudy Munoz case to consider whether I would be an

appropriate medical expert. I received the CT and MRI scans for Noah Whitmer on December 29, 2009, and shortly before that, I received his medical records. On January 4, 2010, I made some notes for the defense attorneys. I also created a PowerPoint document, which included Noah Whitmer's scans and my comments. I informed Mr. Uriarte that I was willing to testify as an expert in the case, and I even offered to do so *pro bono*.

3. Looking back at my notes and PowerPoint, I had determined that the cause of the subdural hematoma and retinal hemorrhage in this case was a venous thrombosis, which is a blood clot in a vein of the brain. The radiologist even described this cortical venous thrombosis in the MRI scan report. The primary issue in Noah Whitmer's case is what caused the venous thrombosis. The doctors in this case made a presumption that because there was a subdural hematoma and retinal hemorrhaging, there must have been violent shaking.

4. I have never seen a case where shaking—however violent—caused a venous thrombosis. There has to be impact trauma to cause a venous thrombosis, and here there is no physical evidence on Noah Whitmer's head or body to suggest impact.

5. Based upon my notes from reviewing the CT and MRI scans of Noah Whitmer, it appears to me that Noah suffered a series of strokes from venous thrombosis, causing some hemorrhaging. There are indications of some blood collections between the brain and the skull. Vinchon, a pediatric neurosurgeon, recently published a paper that explains how preexisting collections from birth can have new hemorrhaging from a venous thrombosis. This was essentially what Dr. Ronald Uscinski testified happened in this case.

6. There is usually a medical cause for venous thrombosis, and the defense attorneys really needed a clinical specialist on the medical side in this case to give a thorough medical explanation. Impact trauma is a pretty obvious cause of venous thrombosis because there tends to be bruising of some kind or a skull fracture.

7. There are, however, non-traumatic triggers as well. Infection, dehydration, or both can be a primary trigger for venous thrombosis, including in infants with a predisposing condition (e.g. thrombophilia , or overclotting condition).

The symptoms of infection may be obvious or subtle. Infants with infection often present a prodrome, or onset of symptoms of disease, as simply fussy, or not eating well, or not behaving as they have in the past. In 5–10% of cases with children being fussy or not eating well, these can be signs that something more serious is occurring. So when we see a venous thrombosis, we often ask if there was anything that looked like an infection.

8. Symptoms of thrombosis may also manifest as very subtle seizures, but parents and caregivers don't always notice these seizures because the child might have staring spells or periods of apnea in which he or she stops breathing.

9. When a baby is not feeding well, taking in too little fluid, and having diarrhea, dehydration decreases the water content in the blood vessels, which causes the cells and proteins to become rigid, which can cause thrombosis.

10. Habeas counsel has provided me with medical records in this case that indicate that:

- Noah Whitmer had a cough and wheezing issues before he was admitted to the hospital on April 20, 2009
- Noah Whitmer's chest x-ray from April 20, 2009 revealed hazy bilateral pulmonary opacities
- Noah Whitmer's sputum tested positive for pneumonia and strep
- Noah Whitmer's respiratory culture collected on April 20, 2009 indicated a heavy growth of staphylococcus aureus and streptococcus pneumoniae
- Noah Whitmer was administered antibiotics
- Noah Whitmer's opthamologist thought that his retinal hemorrhages looked like those produced by meningitis, but Noah was not tested for meningitis

11. These are all powerful indications that Noah Whitmer had an infection when he was admitted to INOVA–Fairfax and that this infection, not trauma, caused the venous thrombosis that is visible on Noah's scans.

12. Meningitis is an infection of the meninges (dura-arachnoid), the membranes between the brain and the skull. The dura-arachnoid becomes inflamed, and inflammation of the membranes can spread to the blood vessels, which can cause subdural hemorrhages, and the subdural hemorrhage can be associated with retinal hemorrhages. The eyes are connected to brain tissue and its blood vessels by the optic nerve and its blood vessels, so anything that happens in the brain can manifest in the eye. When doctors see retinal hemorrhages and presume a child was shaken, they mistakenly treat the eye as a separate organ that has been shaken. Retinal hemorrhages are most often associated with hemorrhages inside the head.

13. A well known complication of meningitis, or other infections, is venous thrombosis, a clotting of blood vessels, which can lead to intracranial hemorrhage and brain injury, including in an infant with a pre-existing condition (e.g. thrombophilia or overclotting condition). Infections leading to meningitis can originate as respiratory infections, sinus infections, ear infections, or other common infections. The fact that Noah didn't have an extremely high fever doesn't mean he didn't have meningitis. The only way to diagnose meningitis definitely is through a lumbar puncture or spinal tap. According to Noah Whitmer's medical records, no such procedure was utilized in this case.

14. Habeas counsel has also provided me with medical records in this case that indicate:

    - Noah Whitmer's parents told medical personnel at INOVA–Fairfax that a "wooden plaque" had fallen on Noah's head approximately ten days prior to his hospitalization.
    - Michael Whitmer—Noah's father—told medical personnel at INOVA – Fairfax that Noah's paternal grandfather had suffered from febrile seizures.
    - Michael Whitmer told medical personnel at INOVA–Fairfax that he had male cousins with muscular dystrophy.
    - Michael Whitmer told medical personnel at INOVA–Fairfax that Noah's mother – Erin Whitmer – had a female relative who died at age eight due to a "chromosomal abnormality."

15. These are all potentially relevant areas of inquiry that should have been investigated by the treating physicians. A wooden plaque falling on an infant's head is an example of accidental impact trauma that can trigger a pre-existing condition (e.g. thrombophilia or overclotting condition).

Unfortunately, the records in this case reveal that the Commonwealth made an immediate and medically inappropriate diagnosis of "shaken baby syndrome." Because the treating physicians presumed abuse, they failed to do any further investigation and ignored some very obvious relevant pieces of information.

16. Michael Whitmer, Noah's father, also indicated to medical personnel that Noah was up-to-date on his immunizations, and Noah's pediatric records confirm that. Presumably, at his 4-month check-up, Noah received vaccinations for:

    - Pneumococcal
    - Measles
    - Rotavirus
    - Diptheria, Tetanus, Pertussis
    - Polio
    - H.Influenzaei type B (HIB)

17. When we see an infant under 6-months-old with venous thrombosis, we automatically start looking back at the mother's pregnancy, labor and delivery, but we also look at any illnesses the child may have had since they were born. Signs of illnesses include feeding problems and colds. However, when an infant is vaccinated, that is essentially giving them a mild infection. Sometimes after an infant has just been vaccinated, they get a fever. However, vaccination may be cause for concern in an infant with a pre-existing condition (e.g. thrombophilia) that might not even be diagnosed. But given Noah Whitmer's acute condition, the possibility that he had some pre-existing condition could not be diagnosed unless medical personnel did a careful and thorough family history.

18. In these types of cases, where there is bleeding in the brain but no external signs of injury, the SBS community wants simple explanations. In fact, these cases are complex and involve many factors. It is critically important for treating medical professionals to consider all explanations and develop differential diagnoses. That never happened in this case. Instead, all of the treating physicians simply assumed trauma and stopped looking for alternative explanations. This is not sound science and cannot be the basis of a reliable prosecution.

19. If trial counsel had obtained a continuance, I would have gladly testified to all of the above, and would have done so even if I could not be paid for my services.

20. In addition, I would have recommended that trial counsel seek the expert assistance of a pediatrician. Such an expert would have been necessary to address the myriad relevant pediatric medical issues in this case.

21. I understand that signing an affidavit is similar to testifying in court. I have carefully reviewed this affidavit before signing it and have been given the opportunity to make any necessary changes to ensure its accuracy.

FURTHER, AFFIANT SAYETH NAUGHT.

Patrick D. Barnes

PATRICK D. BARNES, MD

Signed and sworn before me this 14th day of November, 2012.

_____

NOTARY PUBLIC

My commission expires: _____

Notary registration number: _____

Name: Patrick D. Barnes, M.D. **Aug. 2009**
Office Address: Department of Radiology
Lucile Salter Packard Children's Hospital
Stanford University Medical Center
725 Welch Road
Palo Alto, CA 94304
E-Mail: pbarnes@stanford.edu **Phone:** 650-497-8601
Place of Birth: Oklahoma City, Oklahoma, USA **Fax:** 650-497-8745

Education:

1965-1969	Letters / Pre-Medicine	University of Oklahoma, Norman, OK
1969-1973	Doctor of Medicine	University of Oklahoma College of Medicine, Oklahoma City, OK

Postdoctoral Training:

Residency:

1973-1976 Diagnostic Radiology, University of Oklahoma College of Medicine, Oklahoma City, Oklahoma

Fellowship:

1976-1977 Fellow in Pediatric Neuroradiology and Cardiovascular Radiology, Children's Hospital and Harvard Medical School, Boston, MA

Licensure and Certification:

1973	Federal Licensure Examination Certificate
1974	Oklahoma State Board of Medical Examiners
1977	American Board of Radiology Certificate in Diagnostic Radiology
1986	Commonwealth of Massachusetts Board of Registration in Medicine
2000	Medical Board of California C50437
1995	American Board of Radiology Certificate of Added Qualifications in Neuroradiology
2008	American Board of Radiology Maintenance of Certification in Neuroradiology

Academic Appointments:

1976-1977	Instructor in Radiology, University of Oklahoma College of Medicine
1977-1986	Lecturer in Radiologic Technology, University of Oklahoma College of Health
1977-1982	Assistant Professor of Radiology, University of Oklahoma College of Medicine
1980-1986	Adjunct Faculty, Radiologic Technology, Oscar Rose Junior College
1980-1986	Clinical Assistant Professor of Neurosurgery, University of Oklahoma College of Medicine
1982-1986	Associate Professor of Radiology, University of Oklahoma College of Medicine
1987-1992	Assistant Professor Radiology, Harvard Medical School
1992-2000	Associate Professor of Radiology, Harvard Medical School

2000-	Clinical Associate Professor of Radiology, Stanford University Medical Center
2002-	Associate Professor of Radiology, Stanford University Medical Center
2007-	Professor of Radiology, Stanford University Medical Center

Hospital and Affiliated Institution Appointments:

1977-1986	Pediatric Radiologist, Neuroradiology and Cardiovascular Radiology, Oklahoma Children's Memorial Hospital, Oklahoma City, Oklahoma
1977-1986	Consulting Radiologist, Oklahoma Memorial Hospital and Veterans Administration Hospital, Oklahoma City, Oklahoma
1984-1986	Consulting Radiologist, Oklahoma Diagnostic Imaging Center, Oklahoma City, Oklahoma
1987-1991	Associate Radiologist, Neuroradiology, The Children's Hospital, Boston, MA
1987-2000	Consulting Radiologist, Brigham and Women's Hospital, Beth Israel Hospital, New England Deaconess Hospital, Dana Farber Cancer Institute, Boston, MA
1990-1997	Clinical Coordinator, Magnetic Resonance Imaging, Children's Hospital, Boston, MA
1992-1995	Chief, Section of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1995-1999	Chief, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1995-2000	Board of Directors, Children's Hospital Radiology Foundation, Inc.
1996-2000	Clinical Executive Committee, Department of Radiology, Children's Hospital, Boston, MA
1997-1998	Associate Director of CT, Department of Radiology, Children's Hospital, Boston, MA
1997-1999	Director of MRI, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Director, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Treasurer, Children's Hospital Radiology Foundation, Inc.
1999-2000	Associate Chief for Clinical Operations, Department of Radiology, Children's Hospital, Boston, MA
2000	Senior Associate Neuroradiologist, Department of Radiology, Beth Israel Deaconess Medical Center, and Harvard Medical Faculty Physicians, Inc.
2000-	Staff Physician, Pediatric Neuroradiologist, Lucile Salter Packard Children's Hospital and Stanford University Medical Center
2001-	Interim Director, Pediatric Radiology, Lucile Salter Packard Children's Hospital (Jun-Aug./ JCAHO Survey)
2002-	Chief, Section of Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center, Palo Alto, CA
2002-	Medical Director, MRI/CT Center, Lucile Salter Packard Children's

Hospital

Other Professional Positions and Major Visiting Appointments:

1988	Visiting Professor, The Western Pennsylvania Hospital, Pittsburg, PA
1989	Visiting Professor, New England Medical Center and Tufts University Medical School, Boston, MA
1989	Visiting Professor, Akron Children's Hospital, Akron General Hospital, and Northeastern Ohio Universities College of Medicine, Akron, Ohio
1990	Visiting Professor, Rhode Island Hospital and Brown University College of Medicine, Providence, R.I.

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1991	Visiting Professor, University of Massachusetts Medical Center and Medical School, Worcester, MA
1993	Visiting Professor, Columbus Children's Hospital and the Ohio State University Hospitals, Columbus, OH
1993	Visiting Professor, Christchurch Hospital, University of Otago, Christchurch, New Zealand
1993	Visiting Professor, Royal Children's Hospital, University of Melbourne, Melbourne, Australia
1993	Visiting Professor, Royal Alexandra Hospital for Children, University of Sydney, Sydney, Australia
1993	Visiting Professor, Prince of Wales Children's Hospital, University of New South Wales, Sydney, Australia
1997	Visiting Professor, Montreal Children's Hospital, Montreal General Hospital, Montreal Neurologic Institute, McGill University, Montreal, Quebec, Canada
1998	Visiting Professor, Children's Hospital of Pittsburgh, University of Pittsburgh, Pittsburgh, PA
1998	Visiting Professor, William Beaumont Hospital, Royal Oak, MI
2000	Visiting Professor, Rhode Island Hospital and the Hasbro Children's HospitalBrown University School of Medicine, Providence, RI
2000	Visiting Professor, Massachusetts General Hospital, The Mass General Hospital for Children, and Harvard Medical School, Boston, MA
2008	Visting Professor, Department of Radiology, Duke University Medical Center, Durham NC.

Hospital and Health Care Organization Service Responsibilities:

1977-1986	Staff Pediatric Radiologist and Section Chief, Pediatric Neuroradiology and Cardiovascular Radiology, Oklahoma Children's Memorial Hospital
1987-1992	Associate Radiologist, Neuroradiology, The Children's Hospital, Harvard Medical School, Boston, MA
1992-1995	Chief, Section of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1995-2000	Chief, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA

1997-1998	Associate Director of CT, Department of Radiology, Children's Hospital, Boston, MA
1997-1999	Director of MRI, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Director, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Associate Chief for Clinical Operations, Department of Radiology, Children's Hospital, Boston, MA
2000-	Pediatric Neuroradiologist, Lucile Salter Packard Children's Hospital and Stanford University Medical Center
2001-	Section Chief, Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center
2001-	Interim Director, MRI/CT Center, Lucile Salter Packard Children's Hospital, Stanford University Medical Center
2002-	Interim Director, Pediatric Radiology, Lucile Salter Packard Children's Hospital (Jun-Aug./ JCAHO Survey)
2002-	Chief, Section of Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center, Palo Alto, CA
2002-	Medical Director, MRI/CT Center, Lucile Salter Packard Children's Hospital

Major Administrative Responsibilities:

1984-1986	Clinical Project/Program Consultant, Oklahoma Diagnostic Imaging Center, University of Oklahoma Health Sciences Center, Oklahoma City, Oklahoma
1985-1986	Clinical Project/Program Director, Oklahoma Teaching Hospitals, Magnetic Resonance Center
1987-1990	Clinical Coordinator, The Children's Hospital MRI Determination-Of-Need Process, Department of Public Health, The Commonwealth of Massachusetts, DON Certification, Jan. 1988.
1987-1990	Clinical Coordinator for MRI, The Children's Hospital and The Joint Center for Magnetic Resonance Imaging
1990-1997	Clinical Coordinator, Children's Hospital MRI Service.
1992-1995	Chief, Section of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1992-1999	Co-Director, Combined Neuroradiology Fellowship Program, Brigham & Women's Hospital, Beth Israel Hospital, Children's Hospital, New England Deaconess Hospital, Boston, MA
1992-1999	Director, Pediatric Neuroradiology Fellowship Program, Department of Radiology, Children's Hospital, Boston, MA
1995-2000	Chief, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA

1996-2000	Board of Directors, Children's Hospital Radiology Foundation, Inc (CHRFI), Children's Hospital, Boston, MA
1996-2000	Clinical Executive Committee, Department of Radiology, Children's Hospital, Boston, MA
1997-1998	Associate Director of CT, Department of Radiology, Children's Hospital, Boston, MA
1997-1999	Director of MRI, Department of Radiology, Children's Hospital, Boston, MA
1998-1999	Chair, Bylaws Committee, Children's Hospital Radiology Foundation, Inc (CHRFI), Children's Hospital, Boston, MA
1999-2000	Treasurer, Children's Hospital Radiology Foundation, Inc.
1999-2000	Director, Division of Neuroradiology, Department of Radiology, Children's Hospital, Boston, MA
1999-2000	Associate Chief for Clinical Operations, Department of Radiology, Children's Hospital, Boston, MA
2000-	Pediatric Neuroradiologist, Lucile Salter Packard Children's Hospital and Stanford University Medical Center
2001-	Interim Director, Pediatric Radiology, Lucile Salter Packard Children's Hospital (Jun-Aug./ JCAHO Survey)
2002-	Chief, Section of Pediatric Neuroradiology, Lucile Salter Packard Children's Hospital, Stanford University Medical Center, Palo Alto, CA
2002-	Medical Director, MRI/CT Center, Lucile Salter Packard Children's Hospital

Major Committee Assignments:

Hospital and Medical School:

1977-1981	Safety Committee, Oklahoma Children's Memorial Hospital
1977-1986	Neonatal Care Committee, Oklahoma Children's Memorial Hospital
1977-1986	Utilization Review Committee, Oklahoma Children's Memorial Hospital

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1979-1986	Education and Research Committee, Oklahoma Children's Memorial Hospital
1984-1985	Chairman, State of Oklahoma Teaching Hospitals Task Force on Magnetic Resonance, Oklahoma City, OK
1985-1986	Quality Assurance Committee, Oklahoma Children's Memorial Hospital
1988-1990	Chairman, Joint Center for Magnetic Resonance Imaging, Consortium Clinical and Research Committee, Boston, MA
1988-2000	Pediatric Brain Tumor Working Group, The Children's Hospital and Dana-Farber Cancer Institute, Boston
1988	Steering Committee, Magnetic Resonance Imaging, Department of Radiology, The Children's Hospital, Boston
1989-1991	Chair, Radiology Quality Assurance/Quality Improvement Audit Committee, Children's Hospital, Boston

106.	Radiology Quality Improvement/Risk Management Committee, Children's Hospital, Boston
1992-	Neuroradiology Consultant, Child Protection Service, Children's Hospital, Boston
1992-2000	Department of Radiology Sedation & Contrast Media Committee, Children's Hospital, Boston
1996	Review of the Department of Neurology, Ad Hoc Review Committee, Children's Hospital, Boston
1998-1999	Neuroscience Business Planning Steering Committee and Marketing Team, Children's Hospital, Boston
1998-1999	Harvard Medical School Information Technology Initiative, Hospital and Clinical Linkages Committee, Harvard Medical School and Children's Hospital, Boston
1991-1999	Representative, Department of Radiology, Physician's Leadership Council of the Physician's Organization, Children's Hospital, Boston
2000-	Sedation Committee, Lucile Salter Packard Childrens Hospital at Stanford, Palo Alto, CA
2000-	MR / CT Imaging Facility Planning Committee, Lucile Salter Packard Childrens Hospital at Stanford, Palo Alto, CA
2000-	6-Sigma GEMS MR Capacity Committee, Stanford University Medical Center, Palo Alto, CA.
2005-	Phases I, II LPCH Expansion Committee, Imaging.

Regional:

1985-1986	Consultant on MRI, Oklahoma Health Planning Commission, Technical Advisory Committee, Oklahoma City, OK
2008	Member, Child Abuse Task Force, Lucile Packard Children's Hospital, Stanford University Medical Center, and Santa Clara Valley Medical Center.

National:

1987-1999	Quality Assurance Review Center, National Brain Tumor Committee, and Diagnostic Imaging Committee, Pediatric Oncology Group - High-risk Medulloblastomas, Providence RI
1991-1993	Pediatric Medical Advisory Board for MRI, General Electric Medical Systems.
1991-2000	Member, Neurology Major Test Committee, American Board of Psychiatry and Neurology, National Board of Medical Examiners, Philadelphia, PA

1998	Expert Panel Participant, Evidence-Based Guideline Development for the Management of Children Younger than Two Years of Age with Minor Head Trauma, Packard Foundation.

2000- Expert Panel Participant, Evidence-Based Neuroimaging in the Neonate-Practice Parameter Development Committee, American Academy of Neurology.

2005- Neuroradiologic Consultant / Central Reviewer, Neuroimaging and Neurodevelopmental Outcome, SUPPORT Multicenter Project, Neonatal Research Network, National Institute of Child Health and Human Development (NICHD).

2006- Neuroradiologic Consultant / Central Reviewer, Intervention Trial of Hypothermia for Term HIE Multicenter Project, Neonatal Research Network, National Institute of Child Health and Human Development (NICHD).

2007-2008 Chair, Child Abuse Task Force, Society for Pediatric Radiology.

Professional Societies and Offices:

1977-1986 Oklahoma County Medical Society
1977-1986 Oklahoma State Medical Association
1977-1986 Central Oklahoma Radiological Society
1977-1986 Oklahoma State Radiological Association
1977-1986 Central Oklahoma Pediatric Society
1977-1986 Oklahoma City Clinical Society
1977-1986 Oklahoma Neurological Society
1977- American Medical Association
1977- Radiologic Society of North America
1977- American College of Radiology
1980-1986 Rocky Mountain Neurosurgical Society
1980- Society for Pediatric Radiology
1980- American Society of Neuroradiology
1980- American Roentgen Ray Society
1987- New England Roentgen Ray Society
1987- Boston Neuroradiology Club
1987- Boston Pediatric Radiology Club
1987- Massachusetts Radiological Society
1988-1998 Society of Magnetic Resonance Imaging
1991-1992 Member, Pediatric Neuroradiology Subcommittee on Training and Practice Standards, American Society of Neuroradiology
1991- The Kirkpatrick Society
1992-1996 Chair, Pediatric Neuroradiology Committee, Society for Pediatric Radiology
1992-1998 Chair, Pediatric Neuroradiology Subcommittee on Training and Standards, American Society of Neuroradiology
1992-1993 Co-Founder and member-at-large, Steering Committee, Pediatric Neuroradiology Section of the American Society of Neuroradiology - the American Society of Pediatric Neuroradiology
1993-1995 Member-at-Large, Executive Committee, American Society of Pediatric Neuroradiology, and alternate Representative to Subspecialty Council, American Society of Neuroradiology
1995-1996 Treasurer, American Society of Pediatric Neuroradiology

1996-1997	Secretary and Chair, Membership Committee, American Society of Pediatric Neuroradiology
1996	Chair, Subcommittee "Standard for Cranial Computed Tomography in Infants and Children", The Society for Pediatric Radiology and American College of Radiology

1997	Chair, Subcommittee "Standard for Cranial Magnetic Resonance Imaging in Infants and Children", The Society for Pediatric Radiology and American College of Radiology
1996	Member, Subcommittee "Standard for Sedation/Analgesia in Pediatric Radiology" (M. Cohen, Chair), The Society for Pediatric Radiology and American College of Radiology
1997-1998	Vice President, President-Elect, and Chair, Nominating/Award Committee, American Society of Pediatric Neuroradiology
1998	Member, Caffey Awards Committee, Society for Pediatric Radiology 41st Annual Meeting, Tucson, AZ, May 7-9
1998	Chair, Derek Harwood-Nash Award Committee, American Society of Pediatric Neuroradiology, American Society of Neuroradiology 36th Annual Meeting, Philadelphia, PA, May 17-21
1998-1999	President and Chair, Program/Education Committee, American Society of Pediatric Neuroradiology
1998-1999	Member, Executive Committee, Program Committee, Clinical Practice Committee, Clinical Outcomes Research Committee, American Society of Neuroradiology
1999-2000	Chair, Board of Directors, American Society of Pediatric Neuroradiology
2000-	Chair, Standards and Guidelines Committee, American Society of Pediatric Neuroradiology
2000-	Member, Child Abuse Committee, Society for Pediatric Radiology
2007	Chair, Child Abuse Task Force, Society for Pediatric Radiology
2008	Member, Child Abuse Task Force, Society for Pediatric Radiology
2008	Member, Neuroradiology Committee, Society for Pediatric Radiology

Editorial Boards:

1988-	Reviewer, Radiology (journal of the Radiological Society of North America)
1988-	Reviewer, American Journal of Neuroradiology (journal of the American Society of Neuroradiology)
1991-	Editorial Board, Reviewer, Journal of Child Neurology
1991-	Reviewer, American Journal of Roentgenology (American Roentgen Ray Society)
1993-	Reviewer, Neuroradiology
1993-	Reviewer, Pediatrics
1993-	Reviewer, Journal of Pediatrics
1994-	Editorial Board, Reviewer, Pediatric Radiology (Journal of The Society for Pediatric Radiology and the European Society for Pediatric Radiology)

1995-1997	Associate Editor for Pediatric Neuroradiology, International Medical Image Registry
1995-	Reviewer, Journal of Computed Assisted Tomography
1997-	Reviewer, Neurology

Awards and Honors:

1969	Letzeiser Honor List, University Of Oklahoma
1972	Alpha Omega Alpha
1973	Graduation with Honors, Doctor of Medicine, University of Oklahoma College of Medicine

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1995	Derek Harwood-Nash Outstanding Pediatric Neuroradiology Paper: **Tzika AA**, Barnes PD (mentor), Tarbell NJ, Nelson SJ, Scott RM. "Multivoxel proton spectroscopy of childhood brain tumors", presentation at ASNR 33rd Annual Meeting, Chicago, IL.
1996	Spirit Award, Children's Hospital, Boston, MA.
1996	Honorary Member, Australasian Society of Pediatric Imaging
1997	Kirkpatrick Young Investigator Award: **Alberico RA**, Barnes PD (mentor), Robertson RL, Burrows PE. "Dynamic cerebrovascular imaging in pediatric patients with use of helical CT angiography", paper presentation at the Society for Pediatric Radiology 40th Annual Meeting, St. Louis, MO.
1997	Cum Laude Citation (Scientific Exhibit): **Levine D**, Barnes PD (mentor), Madsen JR, Hulka CA, Li W, Edelman RR. "HASTE MR imaging improves sonographic diagnosis of fetal central nervous system anomalies", scientific exhibit and paper presentation at Radiological Society of North America 83rd Scientific Assembly and Annual Meeting, Chicago, IL.
1998	John A. Kirkpatrick Jr. Teaching Award, Pediatric Radiology Fellowship Program, Department of Radiology, Children's Hospital and Harvard Medical School, Boston, MA.
1999	Derek Harwood-Nash for Outstanding Pediatric Neuroradiology Paper: **Robertson RL**, Ben-Sira L, Schlaug G, Maier SE, Mulkern RV, Duplessis A, Barnes PD (mentor), Robson CD. Line scan diffusion imaging of the brain in neonatal cerebral infarction, paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA.
2000	Medical Intelligence Corporation Scientific Achievement Award for Outstanding Contributions to Neuroimaging in Enhancing Understanding of Timing of Fetal Injury, Las Vegas, Nevada, October 19, 2000.
2000	Outstanding Head & Neck Radiology Paper: **Robson CD**, Mulliken JB, Robertson RL, Proctor MR, Barnes PD (mentor). Prominent basal emissary foramina in syndromic craniosysnostosis – correlation with phenotype and molecular diagnosis, paper presented at the ASNR/ASPNR/ASHNR Annual Meeting, Atlanta, GA, May 2000.
2001	Award of Appreciation for Service & Leadership as Past President 1998-

1999, The American Society of Pediatric Neuroradiology, American Society of Neuroradiology 39th Annual Meeting, Boston, MA, April 23, 2001.

2003 Stanford B. Rossiter Senior Faculty of the Year 2002-2003. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2005 Senior Faculty of the Year 2004-2005. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2006 Senior Faculty of the Year 2005-2006. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2008 The Herman Grossman Lecturer, Department of Radiology, Duke University Medical Center, In Appreciation for Your Contributions to Pediatric Radiology and the Eleventh Annual Herman Grossman Lecturer, April 10, 2008.

RESEARCH, TEACHING, AND CLINICAL CONTRIBUTIONS

Research Activities:

1985 Surface Coil Magnetic Resonance Imaging Clinical Research and Development Project, Dan Galloway, M.D., Patrick Barnes, M.D., and John Prince, Ph.D., Principal Co-Investigators, Oklahoma Diagnostic Imaging Center, University of Oklahoma Health Sciences Center and General Electric Medical Systems, Inc. (IRB#02926).

1986 Magnetic Resonance Imaging and the Evaluation of Morphologic and Biochemical Abnormalities. Patrick Barnes, M.D., and John Prince, Ph.D., Radiology, Principal Co-Investigators, University of Oklahoma Health Sciences Center (IRB#02958), Oklahoma Teaching Hospitals and Philips Medical Systems, Inc. (FDA-PMA-#P840063A).

1987-1991 Pre-Radiation Chemotherapy in the Treatment of Children with Brain Stem Neoplasia, Evaluation with CT and MRI, Pediatric Oncology Group, Cynthia Kretschmer, M.D., The Massachusetts General Hospital, Coordinator (POG8833); Neuroradiologic consultant.

1988-1997 Infant Heart Surgery: CNS Sequelae of Circulatory Arrest, evaluation including Magnetic Resonance Imaging, Jane Newburger, M.D., Principal
1988-1998 Investigator, Department of Cardiology, The Children's Hospital (NIH 1R01HL4178601); Neuroradiologic consultant.

1990-1991 Fast Spin Echo Magnetic Resonance Neuroimaging Project, Patrick Barnes, M.D. and Robert Mulkern, Ph.D., Principal Investigators, Children's Hospital, General Electric Medical Systems, Inc. (CH90-10-099).

1990-1997 Chemotherapy and Radiation Therapy in the Treatment of Seeding Tumors of the CNS in Children, Amy Billett, M.D. and Nancy Tarbell, M.D., Study Chairpersons (DFCI 90-114); Neuroradiologic consultant.

1990-1997	Radiosensitizer Chemotherapy (Etanidazole-SR 2508) and Radiotherapy in Children with Brain Stem Gliomas, Nancy Tarbell, M.D., Study Chairperson (DFCI 90-080); Neuroradiologic consultant.
1991-1999	High Stage Medulloblastomas, Quality Assurance Review Center, Pediatric Oncology Group, Nancy Tarbell, M.D. and Patrick D. Barnes, M.D., Co-Principal Investigators
1992-1997	Stereotactic Radiotherapy for Pediatric Brain Tumors, Nancy Tarbell, M.D., Study Chairperson (DFCI 92-077); Neuroradiologic consultant.
1992-1997	Stereotactic Radiation Therapy for Recurrent or Metastatic CNS Tumors, J. Fontanesi, M.D., J. Loeffler, M.D., P. Barnes, M.D., et al, Coordinators, Pediatric Oncology Group SRS #9373 Protocol.
1994-2000	MR-Techniques in the Assessment of the Newborn Brain, Steven A. Ringer, M.D., Ph.D., Petra S. Huppi, M.D., Co-Principal Investigators, JPN Clinical Research Initiative and Reynolds-Rich-Smith Fellowship; Neuroradiologic Consultant.
1996	Efficacy And Cost-Effectiveness of Fast-Screening Brain MRI Versus Conventional MRI in Children Suspected of Having a Brain Tumor L. Santiago Medina, M.D., Patrick D. Barnes, M.D., A.D. Paltiel, M.D., David Zurakowski, The Society for Pediatric Radiology Research and Education Fund Grant.
1996-2000	Metabolic and Hemodynamic MR Characterization of Pediatric Brain Tumors, A. Aria Tzika, Principal Investigator, Patrick Barnes, M.D., et al, Co-Investigator, American Cancer Society (EDT-80188)
1996-2000	Rehabilitation, Brain Lesions, and Movement in Infants, Edward E. Tronick, Ph.D., Linda Fetter, Ph.D., Alan Leviton, M.D., Co-Principal Investigators (NIH RO1); Neuroradiologic Consultant.
1996-2000	Ultrafast MRI of the Fetal Brain, D. Levine, M.D., Principal Investigator (NIH R29 NS37945-01), Beth Israel Deaconess Medical Center; Neuroradiologic Consultant.

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1999-2000	Pediatric Brain Tumor Consortium, M. Kieran, M.D., Nancy J. Tarbell, M.D. Co-Principal Investigators (NIH/NCI 1 U01 CA 81452-01), Children's Hospital, Massachusetts General Hospital, and Dana Farber Cancer Center; Member, Neuroradiology Committee and Senior Site Neuroradiologic Consultant.
1999-2000	Pediatric Centers for MRI Study of Normal Brain Development, NIH-NINDS-98-13, Michael Rivkin, M.D., principal investigator; Co-investigator and Consultant.
2001-	PAR-98-017 (Reiss) NIMH Longitudinal MRI Study of Brain Development in Fragile X (7.5% effort funded).
2001-	2 R01 MH50047 (Reiss) NIMH Longitudinal Outcomes and Neuroimaging of Fragile X Syndrome (5% effort funded).
2001-	Barth R, MRI of Fetal Ventriculomegaly.
2001	Arriagno R (NIH) Neonatal Diagnosis of Possible Brain Injury in Very

Low Birth Weight Preterm Infants.

2001- Reiss et al. Velocardiofacial syndrome – neuroimaging.

2001- Reiss et al. Bipolar disorder – neuroimaging.

2001- Reiss et al. Coffin-Lowry syndrome – neuroimaging.

2002- Barnes P, et al. Stanford University Certification of Human Subjects Approval IRB Protocal ID 78050: Magnetic Resonance Imaging (MRI) of the Developing Central Nervous System (CNS), March 5, 2002.

2002- Diabetic Ketoacidosis Cerebral Edema Multicenter Study (N. Glaser et al [1% effort funded].

2006- 2U HD 27880-16 Van Meurs (PI). Project period: 04/01/06–03/31/11 NIH/NICHD *Multicenter Network of Neonatal Intensive Care Units Intervention Trial of Hypothermia for Term Hypoxic Ischemic Encephalopathy.* Role: Central MRI reader/Neuroimaging consultant

2006- 2U HD 27880-16 Van Meurs (PI). Project period: 04/01/06–03/31/11 NIH/NICHD *Multicenter Network of Neonatal Intensive Care Units Neuroimaging and Neurodevelopmental Outcome, SUPPORT Multi-Center Project* This project investigates the value of brain magnetic imaging (MRI) in predicting neurodevelopmental outcome in extremely low birthweight (ELBW) infants. Role: Central MRI reader / Neuroimaging consultant

2008 The Well-Nourished and Sleeping Preterm Infant Will Have Improved Brain (Ariagno). Development and Neurodevelopmental Outcome. The Gerber Foundation. Consultant. 08/01/2005-07/31/2008

2008- NIH 1R01 EB008706 Bammer (PI) Project period: 09/01/08 – 08/31/13 Effort: 4.5% ADC: $414,692 "Short Axis EPI MRI at High Field"

2008- Neuroradiologic Consultant / Central Reviewer, National Holoprosencephaly Project, The Carter Center.

Teaching:

Local Contributions:

1976-1979 Course Director and Conference Leader, Pediatric House Staff Core Lecture Series, Pediatric Radiology, Oklahoma Children's Memorial Hospital

1976-1980 Conference Co-leader, Monthly Orthopaedic Radiology-Pathology Conference, Oklahoma Teaching Hospitals

1977-1979 Physician Associates Radiology Lecture Series, College of Allied Health, University of Oklahoma

1977-1982 Conference Co-Leader, Weekly Pediatric Cardiology and Cardiac Surgery Conference

1977-1982 Conference Co-Leader - "Sickle Cell Anemia", Annual Clinical Demonstration for First Year Medical Students, College of Medicine, University of Oklahoma.

1977-1982 Pediatric Cardiac Cine-Angiocardiographic case review and consultation weekly with Pediatric, Pediatric Cardiology, Thoracic Surgery Staff, Residents and Fellows

1977-1985 Pediatric Grand Rounds, Oklahoma Children's Memorial Hospital.

1977-1986	Attending Physician and Conference Leader, Daily and Weekly Clinical Teaching Rounds, Children's Memorial Hospital, University of Oklahoma College of Medicine; Pediatric Radiology Film and Fluoroscopy Review with Radiology, Pediatric, Family Medicine Residents and Medical Students.
1977-1986	Pediatric Neuroradiology Case Review and Consultation daily with Neurosurgery, Neurology, Pediatric, and Adolescent Medicine Staff, Residents, Fellows and Medical Students
1977-1986	Pediatric Computed Tomography, Conventional Tomography, and Special Procedures case review and consultation daily with Pediatric, Pediatric Surgery, Adolescent Medicine, and Orthopedic Staff, Residents, Fellows and Medical Students
1977-1986	Elective Tutorials in Pediatric Neuroradiology and Cardiovascular Radiology for Pediatric, Radiology, Neurosurgery, Neurology and Pediatric Surgery Residents, Fellows, and Students
1977-1986	Weekly Diagnostic Radiology Residency Lecture Series, University of Oklahoma College of Medicine

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1977-1986	Quarterly Radiologic Technology Inservice in Pediatric Neuroradiology and Cardiovascular Radiology Special Procedures
1977-1986	Co-Leader, Weekly Neurosurgery/Neurology Grand Rounds, Oklahoma Teaching Hospitals and St. Anthony Hospital, Oklahoma City, Oklahoma
1978-1982	Course Lecturer, Annual Department of Radiological Sciences Continuing Medical Education Courses, University of Oklahoma Health Sciences Center
1978-1985	Lecturer, Annual Graduate Physics Seminar, College of Allied Health, University of Oklahoma Health Sciences Center
1979-1981	Lecturer, Annual Radiology Grand Rounds, Oklahoma Teaching Hospitals
1980-1985	Lecturer, Pediatric Surgery Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1981-1986	Lecturer, Neurology/Pediatric Neuroradiology Lecture Series, Oklahoma Teaching Hospitals
1982-1985	Participant, Senior Radiology Resident Pre-Board Examinations, University of Oklahoma College of Medicine
1982-1986	Lecturer, Pediatric House Staff Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1983-1986	Course Developer and Director, Resident Final Examination in Pediatric Radiology, University of Oklahoma College of Medicine
1985-1986	Oklahoma Diagnostic Imaging Center Lecture Series, Course Co-Developer and Co-Director
1985-1986	Oklahoma Teaching Hospitals Department of Radiological Sciences, Magnetic Resonance Imaging Lecture Series (Course Developer and Director)

1986	"Magnetic Resonance Imaging for the Referring Physician", Continuing Medical Education Seminar, Program Co-Director, Session Moderator, and Lecturer, Oklahoma Teaching Hospitals and the University of Oklahoma College of Medicine
1987-	Daily Neuroradiology Case Review and Consultation with Pediatric and Adolescent Medicine, Neurology, Neurosurgery, Radiology, Oncology, Radiation Therapy, Orthopedic, ORL/Head and Neck Surgery, Ophthalmology, Plastic Surgery, Oral Surgery, and Neuropathology Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA
1987-	Weekly Pediatric Neurology-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Weekly Pediatric Neurosurgery-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA

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1987-	Weekly Pediatric Neuroncology-Neuroradiology Rounds with Pediatric Oncology, Radiation Oncology, and Neurosurgery Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital and Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Weekly Longwood Medical Area Neuroradiology Conference with Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital, Brigham & Women's Hospital, Beth Israel Hospital, New England Deaconess Hospital, Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Monthly Pediatric ORL/Head & Neck Radiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1987-	Monthly Pediatric Radiology Difficult Case Conference (Risk Management and Quality Improvement) with Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA
1987-	Monthly Boston Area Neuroradiology Club Case Conference with Staff, Fellows, Residents, Medical Students, and visitors (Massachusetts General Hospital)
1987-	Pediatric Neuroradiology Annual Lecture Series, Course Co-Director and Lecturer, for Staff, Fellows, Residents, Medical Students, and visitors.
1987-	Pediatric Neuroradiology Introductory Lectures for Harvard Medical Students and Rotating Radiology Residents, Radiology, Children's Hospital, Boston, MA
1987-1988	Cardiac Radiology Lecture Series, Course Developer and Lecturer, Radiology, Children's Hospital, Boston, MA

1987-1990	Magnetic Resonance Imaging Lecture Series, Course Developer, Director, and Lecturer, Radiology, Children's Hospital, Boston, MA
1987	Invited Lecturer, MRI in Pediatric Neuroradiology, Radiology Grand Rounds, Brigham and Women's Hospital, Boston, MA
1987	Lecturer, "Scoliosis and the Neuroradiologist", "The Impact of MR on Central Nervous System Imaging in Childhood", and "Magnetic Resonance-Diagnostic Imaging Principles", The Children's Hospital and Harvard Medical School Post- Graduate Course, Pediatric Imaging, Boston, MA
1987	Lecturer, "Pediatric Central Nervous System Imaging, The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1988	Invited Lecturer, "MRI in Pediatric Neuroncology", Joint Center for Radiation Therapy Grand Rounds, Children's Hospital, Boston, MA, June 8, 1988
1988	Invited Lecturer, "Magnetic Resonance in Pediatric Imaging", The Children's Hospital and Harvard Medical School Post-graduate Course, Pediatric Medicine

1988	Lecturer, "Magnetic Resonance Imaging of the Pediatric Central Nervous System, Part I - Brain" ; "Magnetic Resonance Imaging of the Pediatric Central Nervous System, Part II – Spine", & Case Review Panel, The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1988	Invited Lecturer, "Magnetic Resonance Imaging", The Children's Hospital, Massachusetts General Hospital, and Harvard Medical School Post-graduate Course, Child Neurology
1989	Lecturer, "Magnetic Resonance in Pediatric Neuroimaging" ; "Magnetic Resonance Imaging in Spinal Dysraphism", The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Boston, MA
1989	Invited Lecturer, "Magnetic Resonance in Pediatric and Adolescent Neuroimaging", The Children's Hospital, Massachusetts General Hospital, and Harvard Medical School Post-graduate Course, Child Neurology
1990	Lecturer, "MR Imaging of the Pediatric Central Nervous System", The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1991	Invited Lecturer, "MRI Signal Patterns-I", & "MRI Signal Patterns-II", Radiology Resident Lecture Series, University of Massachusetts Medical Center and Medical School, Worcester, MA, March 8, 1991
1991	Invited Lecturer, "Pediatric Spine Imaging", Radiology Grand Rounds, University of Massachusetts Medical Center and Medical School, Worcester, MA, March 8, 1991

1991	Invited Lecturer, "MRI of Congenital Spine Lesions", Neurology Grand Rounds, University of Massachusetts Medical Center and Medical School, Worcester, MA, March 9, 1991
1991	Invited Lecturer, "MRI of the Pediatric Central Nervous System", Western Massachusetts Radiological Society, Holyoke, MA, Sept. 24, 1991
1991	Lecturer, "MR Imaging of the Pediatric Central Nervous System", The Brigham & Women's Hospital and Harvard Medical School Post-graduate Course, CT and MRI Update, Cambridge, MA
1991	Invited Lecturer, "MRI in the Pediatric CNS", Harvard Longwood Neurological Training Program Post-graduate Course, Intensive Review of Neurology
1991	Invited Lecturer, "MRI in Pediatrics", Anesthesiology Grand Rounds, Children's Hospital, Boston, MA, Dec. 18, 1991
1992	Invited Lecturer, "Pediatric Brain Tumors", Radiology Grand Rounds, Boston City Hospital, University Hospital, and Boston University Medical School, Boston, MA, Feb. 25, 1992
1991	Invited Lecturer, "Cerebral Dysgenetic Syndromes, Clinical and MRI Correlates", Child Neurology Course, Massachusetts General Hospital, Children's Hospital, and Harvard Medical School Post-Graduate Course, September 1992, Boston, MA

1992	Invited Lecturer, "Pediatric CNS Tumor Imaging", The Harvard Medical School Post-Graduate Course in Neurosurgery-Brain Tumors, November 30, Boston, MA
1993	Invited Lecturer, Massachusetts General Hospital and Harvard Medical School Radiology Review Course, "Congenital CNS Abnormalities". April, Cambridge, MA
1993	Lecturer, "Neuroimaging Techniques in Pediatrics", Child Psychiatry Lecture, Children's Hospital, Boston, MA, June 8,1993
1993	Lecturer, "Neuroimaging in Pediatrics", Radiologic Technologist Inservice Lecture, Children's Hospital, Boston, MA, June 23, 1993
1993	Lecturer, "Neuroimaging-The Pediatric Brain", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Radiology, July 29, Brewster, MA.
1993	Invited Lecturer, "Malformations of the Brain", "Posterior Fossa and Craniocervical Junction Anomalies", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course in Neuroradiology, September 21 and 22, Boston, MA
1994	Lecturer, "Pediatric Neuroimaging: The Brain", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update '94, August 4, New Seabury, MA
1994	Presenter, "Brain Tumors in Children", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course in Neuroradiology, October 3-7, Boston, MA

1994	Lecturer, "Pediatric Brain Imaging", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, Pediatric Brain Imaging, MRI and CT Update, October 27 and 28, Cambridge, MA
1995	Invited Lecturer, "Congenital CNS Abnormalities", Massachusetts General Hospital, Brigham and Women's Hospital, and Harvard Medical School Radiology Review Course, April, Cambridge, MA
1995	Lecturer, "Pediatric Brain Imaging- Protocols and Pitfalls", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update '95, July 26, New Seabury, MA
1995	Invited Lecturer, ""Inflammatory CNS Conditions in Childhood", "Spine and Spinal Cord Anomalies in Childhood", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Neuro MRI, September 19 and 20, Boston, MA
1995	Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI and CT Update, October 12 and 13, Cambridge, MA
1995	Lecturer, "Pediatric CNS Imaging: Protocols & Pitfalls", "Developmental Brain Abnormalities", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI and CT Update, October 12 and 13, Cambridge, MA

1996	Invited Lecturer, "Pediatric Neuroradiology", Massachusetts General Hospital, Brigham and Women's Hospital, and Harvard Medical School Radiology Review Course, April, Cambridge, MA
1996	Moderator, Pediatric Neuroradiology Session, The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update 1996, July 22, Boston, MA
1996	Invited Lecturer, "Imaging of the Orbits and Sinuses: Part I", "Imaging of the Orbits and Sinuses: Part II", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update 1996, July 22, Boston, MA
1996	Invited Lecturer, "Congenital Brain Anomalies" and "Brain Tumors in Children", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Neuro MRI, October 8, Boston, MA
1996	Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update, October 25, Cambridge, MA
1996	Lecturer, "Hydrocephalus", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update, October 25, Cambridge, MA
1996	Invited Lecturer, "Imaging of Cranial and Intracranial Tumors of Childhood", The Brain Tumor Center, Brigham and Women's Hospital,

Children's Hospital, Joint Center of Radiation Therapy, and Dana Farber Cancer Institute, Tumors of the Central Nervous System Post-Graduate Course, November 25, Boston, MA

1997 Invited Lecturer, "Potential Problems and Pitfalls in Pediatric Neuroradiology", Boston University Medical Center, Department of Radiology Grand Rounds, March 20, Boston, MA

1997 Lecturer, "Imaging of Macrocephaly, Parts I and II", The Children's Hospital and Harvard Medical School Post-Graduate Course in Practical Pediatric Imaging: Update 1997, July 21, Boston, MA

1997 Invited Lecturer, "Brain Tumors in the Pediatric Age", and "Congenital and Developmental Conditions of the Spine and Spinal Cord", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Neuro MRI, September 15 and 16, Boston, MA

1997 Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update 1997, October 31, Boston, MA

1997 Lecturer, "Congenital Brain Anomalies--A Problem-Oriented Approach", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI & CT Update 1997, October 31, Boston, MA

1997 Invited Lecturer, "Radiologic Diagnosis of Brain Tumors in Children", Joint Venture Neuroncology, The Partners Health Care System, Dana Farber Cancer Institute, and Harvard Medical School and Brain Tumor Management, November 24, Boston, MA

1997 Moderator, Pediatric Neuroradiology Session, Joint Venture Neuroncology The Partners Health Care System, Dana Farber Cancer Institute, and Harvard Medical School Post-Graduate Course, Tumors of the Central Nervous System and Brain Tumor Management, November 24, Boston, MA

1998 Invited Lecturer, The Brigham & Women's Hospital and Massachusetts General Hospital Radiology Review Post-Graduate Course, "Pediatric Neuroradiology", April 6, Cambridge, MA

1998 Invited Lecturer, "Congenital and Developmental Conditions of the Spine and Spinal Cord", The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course, Basic and Current Concepts in Neuroradiology, Head & Neck Radiology, and Clinical Functional MRI and Spectroscopy, September 16, Boston, MA

1998 Moderator, Pediatric Neuroradiology Session, The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI/CT Update 1998,October 30, Boston, MA

1998 Lecturer, "Major Congenital Brain Anomalies", The Brigham and Women's Hospital and Harvard Medical School Post-Graduate Course, MRI/CT Update 1998, October 30, Boston, MA

1999	Invited Lecturer, "Neonatal MRI: New Techniques", Division of Newborn Medicine Clinical Conferences, Children's Hospital, January 4, Boston, MA
1999	Invited Speaker, Imaging of Brain Tumors in Children, Parents Workshop, Jimmy Fund Clinic, Dana-Faerber Cancer Institute, May 1, Boston, MA.
1999	Invited Speaker, Radiologic Diagnosis of Brain Tumors in Children, Tumors of the Central Nervous System: Management of Brain Tumors Post-graduate Course, Brigham and Women's Hospital, Massachusetts General Hospital, Children's Hospital, Dana-Faerber Cancer Institute, Harvard Medical School, September 13, Boston, MA
1999	Invited Speaker, Congenital and Developmental Conditions of the Spine and Spinal Cord, Neuroradiology, Head & Neck Radiology, Clinical Functional MRI and Spectroscopy Post-graduate Course, Massachusetts General Hospital, Massachusetts Eye & Ear Infirmary, Harvard Medical School, October 6, Boston, MA
1999	Invited Speaker, Potential Pitfalls in Pediatric Neuroradiology, and Session Moderator, Pediatric Neuroradiology Session, MRI/CT Update Post-graduate Course, Brigham & Women's Hospital, Harvard Medical School, October 29, Boston, MA

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2000	Invited Discussant, Pediatric Neuroncology, Neurosurgery, and Neurology Conferences, Department of Radiology, Massachusetts General Hospital, Jan.-Feb., Boston, MA
2000	Basic Technical and Biological Principles of Magnetic Resonance Imaging Lecture Series, Department of Radiology, Beth Israel Deaconess Medical Center, Feb.-May, Boston, MA
2000	Pediatric Neuroradiology Resident Pre-Board Review, Department of Radiology, Beth Israel Deaconess Medical Center, May, Boston, MA
2000-	Daily Pediatric Neuroradiology and Head & Neck CT and MRI Case Review / Consultations with Fellows, Residents, Medical Students, and Visiting Physicians, Lucile Salter Packard Children's Hospital and Stanford University Medical Center, Palo Alto, CA
2000-	Conference Co-Leader, Weekly Pediatric Neuroncology Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo Alto,
2000-	Conference Leader, Weekly Pediatric Neuroradiology, Neurology, and Neurosurgery Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo Alto, CA
2000-	Pediatric Neuroradiology Lectures, Neuroradiology Lecture Series, Department of Radiology, Stanford University Medical Center, Palo Alto, CA
2000-	Faculty Participant, Weekly Neuroradiology Case Review / QI Conference Department of Radiology, Stanford University Medical Center, Palo Alto, CA

2000-	Faculty Participant, Weekly Neurology Case Conference, Stanford University Medical Center, Palo Alto, CA
2000-	Faculty Participant, Weekly Perinatal Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo, Alto, CA
2000	Invited Lecturer, Pitfalls in Pediatric Neuroradiology, Neurosurgery Grand Rounds, Stanford University Medical Center, Palo Alto, CA Sept. 1, 2000
2000-	Faculty Participant, International Perinatal Teleconferences (Hong Kong), Lucile Salter Packard Children's Hospital at Stanford, Palo Alto, CA
2000	Medical Student Clerkship Lecture, Pediatric Neuroradiology, Department of Radiology, Stanford University Medical Center, Palo, Alto, CA, Oct. 12, 2000
2000	Invited Lecturer, Imaging of Neonatal Encephalopathy, Neonatal Intensive Care Clinical Research Conference, Lucile Salter Packard Children's Hospital at Stanford, Palo Alto, CA, Oct. 16, 2000.
2001	Invited Lecturer, Potential Pitfalls in Pediatric Neuroradiology-The Impact of Advancing Neuroimaging Techniques, Department of Radiology, Stanford University Medical Center, Palo Alto, CA, Feb. 13, 2001.
2001	Faculty participant, Weekly Epilepsy Conference, Stanford University Medical Center, Palo Alto, CA.
2001-	Monthly Pediatric Neuroradiology Lecture Series for Neurology Residents & Fellows, Stanford University Medical Center, Palo Alto, CA.
2001-	Monthly Pediatric Neuroradiology Lecture Series for Neurosurgery Residents and Fellows Stanford University Medical Center, Palo Alto, CA.
2001-	Monthly Pediatic Head & Neck Imaging Lecture Series for ORL/Head & Neck Residents and Fellows, Stanford University Medical Center, Palo Alto, CA.
2001-	Pediatric Neuroradiology Lectures, Pediatric Radiology Lecture Series, Department of Radiology, Stanford University Medical Center, Palo Alto, CA.

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Regional, national, or international contributions:

1988	Invited Lecturer, "Neurocutaneous Syndromes", & "Pediatric Spine Imaging-Spinal dysraphism", Western Pennsylvania Hospital, Pittsburg, PA, Nov. 3, 1988
1989	Invited Lecturer, "Pediatric Spine Imaging", New England Medical Center and Tufts Medical School, Feb. 9, 1989
1989	Invited Lecturer, "MRI-Basic Principles and Pediatric Applications", Akron Children's Hospital, Akron, OH, May 3, 1989
1989	Invited Lecturer, "MRI in Pediatric Spine Imaging", Northeast Ohio University Medical Center, Akron, OH, May 3, 1989

1989	Invited Lecturer, "MRI in Pediatric and Adolescent Neuroimaging", Akron Radiological Society, Akron, OH, May 3, 1989
1989	Invited Discussant, Neuroimaging-Neuropathology Correlation Conference, Akron Children's Hospital, Akron, OH, May 4, 1989
1989	Invited Lecturer, "Imaging of the Neurocutaneous Syndromes", Akron Children's Hospital, Akron, OH, May 4, 1989
1990	Invited Lecturer, "MRI in Pediatric Neuroimaging-Guidelines", & "Pediatric Spine Imaging", Rhode Island Hospital and Brown University Medical School, April 2, 1990
1990	Invited Lecturer, " Neuroimaging of the Neurocutaneous Syndromes", Radiology Grand Rounds, Rhode Island Hospital and Brown University Medical School, April 2, 1990
1991	Moderator, Pediatric Neuroradiology, Special Scientific Session, American Society of Neuroradiology, 29th Annual Meeting, Washington, D.C.
1991	Moderator and Discussant, Pediatric Neuroradiology Scientific Session, Radiological Society of North America 77th Annual Meeting, Chicago
1992	Invited Lecturer, "Signal Intensity Patterns in MRI of the Pediatric CNS", Radiology Resident Lecture, Ohio State University Health Sciences Center, Columbus, OH, April 8, 1992
1992	Invited Lecturer, "MRI in Pediatric CNS Imaging", Columbus Radiological Society, Columbus, OH, April 8, 1992
1991	Invited Lecturer, "Pediatric Spine Imaging", Radiology Grand Rounds, Columbus Children's Hospital, Columbus, OH, April 9, 1992
1992	Co-Moderator and Discussant, Scientific Session on Pediatric Neuroradiology, Society for Pediatric Radiology 35th Annual Meeting, May 17, Orlando, FL
1992	Invited Lecturer and Panelist, "Sedation in Pediatric Neuroradiology", American Society of Neuroradiology 30th Annual Meeting, June 3, St. Louis, MO
1992	Panelist, Scientific Session on Pediatric Neuroradiology, American Society of Neuroradiology 30th Annual Meeting, June 3, St. Louis, MO

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1993	Co-Moderator and Co-Discussant, Neuroradiology Long Papers Session, Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 13, 1993
1993	Co-Discussant, Pediatric Scientific Session, American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 19, 1993
1993	Discussant, Pediatric Specialties Scientific Session, American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 19, 1993

1993	Invited Lecturer, "MRI in Pediatric Imaging", Christchurch Hospital, University of Otago, Christchurch, New Zealand, Oct. 4, 1993
1993	Invited Lecturer, "Basics of MRI", & "Signal Intensity Patterns in MRI of the Pediatric CNS", and Discussant, Epilepsy Conference, Royal Children's Hospital, University of Melbourne, Melbourne, Australia, Oct. 11, 1993
1993	Invited Lecturer, "MRI in Pediatric Cerebrovascular Disease", and Discussant, Pediatric Neurology and Neurosurgery Conference, Prince of Wales Hospital, University of Sydney, Sydney, New South Wales, Australia, Oct. 13, 1993
1993	Invited Discussant, Radiology Resident Case Review Lecture, Royal Alexandra Hospital for Children, University of Sydney, Sydney, New South Wales, Australia, Oct. 13, 1993
1993	Invited Lecturer, " Imaging in Pediatric Neuroncology", "Neurocutaneous Syndromes", "Pediatric Neurovascular Diseases", Australasian Society for Paediatric Imaging (ASPI), October 15-17, Leura, New South Wales, Australia.
1993	Invited Lecturer, "Congenital & Developmental Brain Abnormalities", "Intracranial Inflammatory Processes", "Metabolic and Neurodegenerative Disorders", "Vascular Diseases and Trauma", "Cranial and Intracranial Tumors", "Neurocutaneous Syndromes", "Developmental and Acquired Abnormalities of the Spine and Spinal Neuraxis". ASPI MRI Symposium, October 18, Leura, New South Wales, Australia
1994	Invited Lecturer, "Imaging of the Pediatric Central Nervous System: Current Concepts", The Denby Bowdler Lecture, The Annual Post-Graduate Meeting, The Royal Alexandra Hospital for Children, Sydney, New South Wales, Australia, Oct. 21, 1993
1994	Moderator and Invited Lecturer, Update Course in Pediatric Radiology-Neuroradiology, Radiologic Society of North America, November 28, Chicago, IL.
1995	Invited Lecturer, Current Concepts in Pediatric Imaging-Neuroradiology, The Society for Pediatric Radiology, April 27, Colorado Springs, CO.
1995	Invited Lecturer, Society of Magnetic Resonance Technologists, Pediatric MRI-Sedation and Monitoring, 1994 Annual Regional Meeting, October 8, Boston, MA

1995	Moderator and Invited Lecturer, Update Course in Pediatric Radiology-Neuroradiology, Radiological Society of North America, November 27, Chicago, IL
1995	Co-Moderator and Co-Discussant, Pediatric Scientific Session, American Society of Neuroradiology 33rd Annual Meeting, April 23, Chicago, IL
1995	Co-Moderator and Co-Discussant, Neuroradiology Scientific Session, Society for Pediatric Radiology, 38th Annual Meeting, April 29, Washington, D.C.

1995	Invited Lecturer, Emergency Pediatric Radiology Categorical Course- "Increased Intracranial Pressure"-American Roentgen Ray Society 95th Annual Meeting, April 30, Washington, D.C.
1995	Invited Lecturer, Update Course in Clinical Neuroradiology: Pediatric Neurovascular Imaging, Refresher Course, Radiological Society of North America, 81st Annual Meeting, November 29, Chicago, IL
1995	Invited Lecturer, Special Focus Session: Pediatric Sedation. Radiological Society of North America, 81st Annual Meeting, November 30, Chicago, IL
1996	Co-Moderator, and Co-Director, Pediatric Neuroradiology Session, IPR '96 Pediatric Neuroimaging Symposium, International Pediatric Radiology 3rd Conjoint Meeting, SPR, ESPNR, ASPI, May 25, Boston, MA
1996	Invited Lecturer, "Current and New Concepts in Imaging of the Pediatric Spine" IPR 96 Pediatric Neuroimaging Symposium., International Pediatric Radiology 3rd Conjoint Meeting, SPR, ESPNR, ASPI, May 25, Boston, MA
1997	Invited Lecturer, "Imaging of Head and Neck Masses in Childhood", McGill University, Department of Diagnostic Radiology Grand Rounds, January 20, Montreal, Quebec, Canada
1997	Invited Lecturer, "Cranial and Intracranial Tumors of Childhood: An Overview", Montreal Children's Hospital, Department of Diagnostic Imaging, January 21, Montreal, Quebec, Canada
1997	The Dr. Bernadette Nogrady Lecturer, "Imaging of the Neurocutaneous Syndromes in Childhood", Medical Grand Rounds, Montreal Children's Hospital, McGill University, Jan. 21, Montreal, Quebec, Canada.
1997	Invited Lecturer, "Congenital Malformations of the Brain", Practical MRI Categorical Course, American Roentgen Ray Society, 97th Annual Meeting, May 4, Boston, MA.
1997	Invited Lecturer, "MRI and Other Advanced Imaging Techniques", Spinal Dysraphism Workshop, Society for Pediatric Radiology, May 15, St.Louis, MO.
1997	Invited Lecturer, "Advanced Techniques in Pediatric Neuroradiology", New England Conference of Radiologic Technologists and New England Chapter of the American Radiology Nurses Association 39th Annual Fall Symposium, September 26, Sturbridge, MA

1998	Invited Lecturer, "Imaging of the Pediatric Spine, Part I", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 9, Pittsburgh, PA
1998	Invited Lecturer, "Potential Pitfalls in Imaging of the Pediatric CNS", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 9, Pittsburgh, PA

1998 Invited Lecturer, Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, Teaching Session with Residents and Fellows, February 9, Pittsburgh, PA

1998 Invited Lecturer, "Imaging of the Pediatric Spine, Part II", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 10, Pittsburgh, PA

1998 Invited Lecturer, "Imaging of CNS Injury in Child Abuse", Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, February 10, Pittsburgh, PA

1998 Invited Lecturer, Department of Radiology, Children's Hospital of Pittsburgh and University of Pittsburgh Medical Center, Teaching Session with Residents and Fellows, February 10, Pittsburgh, PA

1998 Invited Lecturer, "Potential Pitfalls in Imaging of the Pediatric CNS", Department of Radiology, William Beaumont Hospital, March 18, Royal Oak, MI

1998 Invited Lecturer, "Imaging of CNS Injury in Child Abuse", Department of Radiology, William Beaumont Hospital, March 18, Royal Oak, MI

1998 Course Director and Moderator, Multimodality Imaging of Head & Neck Lesions in Childhood -- The Oral Cavity, Jaw, and Neck; The Eye and Orbit; The Ear and Temporal bone; The Nose, Paranasal Sinuses, and Craniofacial Structures; Sunrise Sessions, The Society for Pediatric Radiology, 41st Annual Meeting, May 7-9, Tucson, AZ

1998 Co-Moderator, Scientific Session VI--Neuroradiology, The Society for Pediatric Radiology, 41st Annual Meeting, May 9, Tucson, AZ

1998 Invited Lecturer, Focus Session: Scoliosis "Imaging the Spine in Scoliosis", the American Society of Neuroradiology, 36th Annual Meeting, May 17-21, Philadelphia, PA

1998 Course Director and Moderator, Minicourse in Pediatric Neuroradiology: Session I: "Pediatric Neurovascular Diseases"; Session II: "Pediatric CNS Tumors"; Session III: "Congenital and Developmental Abnormalities"; Session IV: "Traumatic, Inflammatory, and Neurodegenerative Diseases", Radiological Society of North America, 84th Scientific Assembly and Annual Meeting, November 29-December 1, Chicago, IL

1998 Invited Speaker, Minicourse in Pediatric Neuroradiology, "Tumors about the Third Ventricle", Radiological Society of North America, 84th Scientific Assembly and Annual Meeting, November 30, Chicago, IL

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1998 Invited Speaker, Special Focus Session--Child Abuse Revisited, Radiological Society of North America, 84th Scientific Assembly and Annual Meeting, December 1, Chicago, IL

1998 Invited Lecturer, "Potential Pitfalls in Imaging of the Pediatric CNS", The Roger A. Hyman Memorial Lecture, Long Island Radiological Society and Winthrop-University Hospital , Dec. 8, Long Island, NY

1999	Invited Speaker, "Shaken Baby Syndrome", Current Issues in Emergency Practice, Seventh Annual Massachusetts Emergency Nurses Association and Massachusetts College of Emergency Physicians Course, April 13, Marlboro, MA
1999	Invited Speaker, "The Pediatric Radiologist as Expert Witness: How I do it", Society for Pediatric Radiology, Postgraduate Course, May 12, Vancouver, B.C., Canada
1999	Pediatric Focus Sessions Director and Moderator, Session I: "Diagnosis and Management of Head and Neck Vascular Anomalies of Childhood"; Session II: "Diagnosis and Management of Craniofacial Anomalies"; Session III: "Diagnosis and Management of Craniocervical Anomalies"; Session IV: Basic Science/Applications – Watershed Patterns: Anatomy and Pathology; Session V: Diagnosis and Management of Pediatric Neuroendocrine Disorders"; Session VI: "Diagnosis and Management of Pediatric Epilepsy", American Society of Neuroradiology/American Society of Pediatric Neuroradiology Annual Meeting, May 22-23, San Diego, CA
1999	Invited Speaker, Neuroncologic Imaging in Children, Neuroimaging Session, Frontiers of Hope, A Brain Tumor Symposium for Patients, Survivors, Family, Friends, and Professionals, The Brain Tumor Society, November 13, Providence, RI
2000	Invited Speaker, Potential Pitfalls in Pediatric Neuroradiology, Parts I & II, Department of Diagnostic Imaging Grand Rounds, Brown University School of Medicine, Rhode Island Hospital, and the Hasbro Children's Hospital, Providence RI.
2000	Invited Speaker, Neuroradiology of Pediatric Scoliosis, Practical Spine Imaging & Image Guided Therapy Symposium, The American Society of Spine Radiology, February 23, Marco Island, FL
2000	Invited Speaker, Diffusion Imaging in Children, ASNR 2000: Advanced Imaging Symposium, American Society of Neuroradiology, April 2, Atlanta, GA
2000	Moderator, Pediatric Scientific Session, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, April 2-8, Atlanta, GA

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2000	Invited Speaker, Pediatric Neuroradiology, Advanced Medical Malpractice Seminar, Office of Legal Education, Executive Office for U.S. Attorneys, United States Department of Justice, May 2, Columbia, SC.
2000	Invited Speaker, Course Director, Syllabus Editor / Co-author, & Session Moderator, Problem-Focused Strategies in Pediatric Neuroradiology: An Interactive Symposium, Society for Pediatric Radiology and American Society of Pediatric Neuroradiology Joint Post-graduate Course, May 4-6,

Naples, FL.

2000 Invited Speaker and Participant, Fetal & Neonatal Neurologic Injury, Part I - Neuroimaging Patterns and the Timing of Fetal Brain Injury – Medical Intelligence Corporation Keynote Addrress; Part II - The Neuroimaging Expert, Birth Injury and the Law VII, Oct. 19, Las Vegas, NV

2001 Invited Speaker and Participant, Imaging of Fetal & Neonatal CNS Injury Parts I-III, 17th Annual Conference on Obstretics, Gynecology, Perinatal Medicine, Neonatalogy, and the Law, Jan. 2-5, San Juan, PR

2001 Invited Speaker, Pediatric Spine Imaging, Fetal and Infant Neuro-MR, Pediatric Brain Imaging I-II, MR Update 2001, Neuroradiology and Musculoskeletal Imaging Advances, Stanford Radiology, Feb. 16, Las Vegas, Nevada

2001 Invited Speaker and Participant, Sam Hersch Cerebral Palsy Symposium at the Salk Institute, Feb. 27-28, La Jolla, CA.

2001 Invited Speaker & Session Co-coordinator, RSNA Oncodiagnosis Panel-Pediatric Brain Tumors, Radiologic Society of North America 87th Scientific Assembly and Annual Meeting, Chicago, IL, Dec. 28, 2001.

2002 Barnes PD. Invited Speaker. Current and Advanced Techniques in Imaging of the Pediatric Central Nervous System. Department of Neurology Grand Rounds. Stanford University Medical Center, Palo Alto, CA, Jan. 30, 2002.

2002 Invited Speaker. Current and Advanced Techniques in Pediatric Otolaryngology / Head & Neck Imaging – A Problem-focused Approach, Western Society of Pediatric Otolaryngology Annual Meeting, Lucile Packard Children's Hospital at Stanford, Palo Alto, CA, Mar. 16, 2002

2002 Invited Speaker. Neuroimaging of congenital and neonatal Infections. Postgraduate Course: Perinatal and neonatal imaging, Society for Pediatric Radiology, Philadelphia, PA, May 2, 2002.

2002 Session Co-Moderator. White Matter Symposium. American Society of Neuroradiology / American Society of Pediatric Neuroradiology, Vancouver, B.C., May 16, 2002.

2003 Barnes PD. Current and Advanced Imaging of the Fetal and Neonatal CNS. Mid-Coastal California Perinatal Outreach Program, 23rd Annual Meeting, Stanford University Schoolof Medicine, Monterey, CA, Jan. 2003.

2003 Barnes PD. Neuroimaging: a medical perspective. Litigating catastrophically injured infant cases, Association of Trial Lawyers of America, Feb.22, 2003, Atlanta, GA.

2003 Barnes PD. Trauma, including Child Abuse. CT & MRI: State of the Art & Unanswered Questions, SPR Postgraduate Course, San Francisco, CA, May 6, 2003.

2004 Barnes PD. Nonaccidental Head Injury in Children. Neurosciences Grand Rounds. Santa Clara Valley Medical Center. San Jose, CA, Feb. 5, 2004.

2004 Barnes PD. Forensic Science, Evidence-based Medicine, and the "Shaken Baby Syndrome": Radiographic Imaging and Findings. American

Academy of Forensic Sciences Annual Meeting, Dallas,Tx, Feb. 16, 2004.

2004 Barnes PD. Nonaccidental Injury of the Developing Brain: Issues, Controversies, and the Mimics. Moderator and Speaker. Neuroimaging Aspects. Focus Session, American Society of Pediatric Neuroradiology. American Society of Neuroradiology Annual Meeting, Seattle, WA, June 7, 2004.

2004 Barnes PD. Co-Moderator, Pediatric scientific session, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, Seattle, WA, June 8, 2004.

2004 Barnes PD. Moderator, Pediatric Session and Speaker. MDCT applications in Pediatric Neuroradiology (Brain, Spine, Head & Neck). 6th Annual International Symposium on Multidetector-Row CT. Stanford University Medical Center, San Francisco CA, June 23, 2004.

2004 Barnes PD. Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. 12th Annual Pediatric Update, Lucille Packard Children's Hospital and Stanford University Medical Center, July 16, 2004.

2005 Barnes PD. Neuroimaging of the pediatric spine – scoliosis. Neuroscience Grand Rounds. Santa Clara Valley Medical Center. San Jose, CA. March 3, 2005.

2005 Barnes PD. Diagnostic imaging of neonatal brain injury. California Association of Neonatologists (CAN) and American Academy of Pediatrics (AAP) District IX Section on Perinatal Pediatrics, 11th Annual Conference, Current Topics and Controversies in Perinatal and Neonatal Medicine, Coronado CA, March 6, 2005.

2005 Barnes PD. Co-moderator, Neuroradiology scientific session, Society for Pediatric Radiology Annual Meeting, New Orleans, LA, May 7, 2005.

2005 Barnes PD. Moderator, CAQ Review Sessions, Pediatric Brain, Head & Neck, and Spine Imaging, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, Toronto, Ontario, Canada, May 26-27, 2005.

2005 Barnes PD. Co-Moderator, Pediatric scientific session, American Society of Pediatric Neuroradiology, American Society of Neuroradiology Annual Meeting, Toronto, Ontario, Canada, May 26, 2005.

2005 Barnes P.Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. 13th Annual Pediatric Update, Lucile Packard Children's Hospital and Stanford University Medical Center, July 8, 2005.

2005 Barnes P.Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. Neurosurgery Grand Rounds, Stanford University Medical Center, July 15, 2005.

2006 Barnes P. Imaging of the Pediatric Central Nervous System and Head & Neck: MRI, CT, US, Nuclear Medicine – Which to do? 14th Annual Pediatric Update, Lucile Packard Children's Hospital and Stanford University Medical Center, July 21, 2006.

2006	Barnes P. Child Abuse: Issues and Controversies in the Era of Evidence-Based Medicine. Pediatric Grand Rounds, Lucile Packard Children's Hospital and Stanford University Medical Center, October 13, 2006.
2006	Hahn J, Barnes P. Prenatal Neurologic Consultations and Management of Brain Malformations. Pediatric Grand Rounds, Lucile Packard Children's Hospital and Stanford University Medical Center, Nov. 3, 2006.
2007.	Barnes PD. Co-Director and Co-Moderator. Brain, Head & Neck, and Spine Imaging. Advances in Pediatric CT and MRI. Department of Radiology, Stanford School of Medicine Postgraduate Course. Las Vegas, Nevada, March 17, 2007.
2007	Barnes PD. Lecturer. Advances in Pediatric CT and MRI: Head & Neck Imaging I (Orbit, Sinus, Ear), Head & Neck Imaging II (Face & Neck), Spine Imaging I (Developmental Anomalies), Spine Imaging II (Acquired Conditions), Brain Imaging III (Acute neurologic conditions – Trauma [including child abuse], hemorrhage, vascular disease), Brain Imaging V (Subacute neurologic conditions – Tumors, epilepsy). Department of Radiology, Stanford School of Medicine Postgraduate Course. Las Vegas, Nevada, March 17, 2007. Course Syllabus.
2007	Barnes PD. Lecturer. How I do it – Advanced Neuro-MRI of Nonaccidental CNS injury and its Mimics. Society for Pediatric Radiology 50th Annual Meeting and Postgraduate Course. Miami FL. April 20, 2007.
2007	Barnes P. Lecturer. Child Abuse: Pitfalls in Pediatric Neuroimaging. EBMS Symposium: An Evidence-based Analysis of Infant Brain and Skeletal Injury. Chicago IL, May 10, 2007.
2007	Barnes P. Lecturer. Child Abuse: Issues and Controversies in the Era of Evidence-Based Medicine. Department of Social Services and Child Protection, Lucile Packard Children's Hospital and Stanford University Medical Center, June 21, 2007.
2007	Barnes P. Lecturer. Child Abuse: Issues & Controversies. Pediatrics CME Program. Salinas Valley Memorial Healthcare System, Salinas CA, Nov. 16, 2007.
2008	Barnes P. Lecturer. Child Abuse and the Mimics. Imaging of Brain, Blood, & Bones. Death of a Child Symposium. The Center for American and International Law. Plano TX, March 4, 2008.
2008	Barnes P. Imaging of Child Abuse: Controversies in the Era of Evidence-Based Medicine. Herman Grossman Visiting Lecturer. Radiology & Pediatrics Grand Rounds. Duke University Medical Center, Durham NC, April 10, 2008.
2008	Barnes P. Update on Brain Imaging in Nonaccidental Trauma. Neuroimaging I Session, Pediatric Radiology Series. Radiologic Society of North America, Chicago IL, Nov. 30, 2008.
2008	Barnes P. Co-Moderator & Discussant, Neuroimaging I Scientific Paper Session, Pediatric Radiology Series , Radiologic Society of North America, Chicago, IL Nov. 30, 2008
2008	Barnes P. Neuroimaging in the Evaluation of Pattern and Timing of Fetal and Neonatal Brain Injury. Fetal & Neonatal Annual Care Conference.

Santa Clara Valley Medical Center. San Jose CA, November 7, 2008.

2009 Barnes P. Medical Imaging in Brain Trauma; Intracranial Hemorrhage and Thrombosis (Krasnokutsky M): Imaging & Pitfalls. An Evidence-based Analysis of Infant Brain & Skeletal Trauma. EBMS Symposium, Denver CO, February 22, 2009.

2009 Barnes P. Imaging of Child Abuse and the Mimics: Controversies in the Era of Evidence-Based Medicine. Innocence Network Conference. South Texas College of Law, Houston TX, March 21. 2009.

2009 Barnes P. Neuroimaging in the Evaluation of Pattern and Timing of Fetal and Neonatal Brain Abnormalities. The Latest Tools and Science to Determine the Origin and Timing of Irreversible Brain Damage. Obstetric Malpractice West Coast Conference & Workshop. San Francisco CA, April 28, 2009.

Teaching Awards:

1998 John A. Kirkpatrick Jr. Teaching Award, Pediatric Radiology Fellowship Program, Department of Radiology, Children's Hospital and Harvard Medical School, Boston, MA.

2003 Stanford B. Rossiter Senior Faculty of the Year 2002-2003. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2005 Senior Faculty of the Year 2004-2005 Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

2006 Senior Faculty of the Year 2005-2006. Outstanding Contributions to Resident Education, Compassionate Patient Care, and Research, Department of Radiology, Stanford University Medical Center.

Major Curriculum and Educational Programs Developed:

1976-1979 Course Director and Conference Leader, Pediatric House Staff Core Lecture Series, Pediatric Radiology, Oklahoma Children's Memorial Hospital

1976-1980 Conference Co-leader, Monthly Orthopaedic Radiology-Pathology Conference, Oklahoma Teaching Hospitals

1977-1979 Physician Associates Radiology Lecture Series, College of Allied Health, University of Oklahoma

1977-1982 Conference Co-Leader, Weekly Pediatric Cardiology and Cardiac Surgery Conference

1977-1982 Conference Co-Leader - "Sickle Cell Anemia", Annual Clinical Demonstration for First Year Medical Students, College of Medicine, University of Oklahoma.

1977-1982 Pediatric Cardiac Cine-Angiocardiographic case review and consultation weekly with Pediatric, Pediatric Cardiology, Thoracic Surgery Staff, Residents and Fellows

1977-1985 Pediatric Grand Rounds, Oklahoma Children's Memorial Hospital.

1977-1986	Attending Physician and Conference Leader, Daily and Weekly Clinical Teaching Rounds, Children's Memorial Hospital, University of Oklahoma College of Medicine; Pediatric Radiology Film and Fluoroscopy Review with Radiology, Pediatric, Family Medicine Residents and Medical Students.
1977-1986	Pediatric Neuroradiology Case Review and Consultation daily with Neurosurgery, Neurology, Pediatric, and Adolescent Medicine Staff, Residents, Fellows and Medical Students
1977-1986	Pediatric Computed Tomography, Conventional Tomography, and Special Procedures case review and consultation daily with Pediatric, Pediatric Surgery, Adolescent Medicine, and Orthopedic Staff, Residents, Fellows and Medical Students
1977-1986	Elective Tutorials in Pediatric Neuroradiology and Cardiovascular Radiology for Pediatric, Radiology, Neurosurgery, Neurology and Pediatric Surgery Residents, Fellows, and Students
1977-1986	Weekly Diagnostic Radiology Residency Lecture Series, University of Oklahoma College of Medicine
1977-1986	Quarterly Radiologic Technology Inservice in Pediatric Neuroradiology and Cardiovascular Radiology Special Procedures
1977-1986	Co-Leader, Weekly Neurosurgery/Neurology Grand Rounds, Oklahoma Teaching Hospitals and St. Anthony Hospital, Oklahoma City, Oklahoma
1978-1982	Course Lecturer, Annual Department of Radiological Sciences Continuing Medical Education Courses, University of Oklahoma Health Sciences Center
1978-1985	Lecturer, Annual Graduate Physics Seminar, College of Allied Health, University of Oklahoma Health Sciences Center
1979-1981	Lecturer, Annual Radiology Grand Rounds, Oklahoma Teaching Hospitals
1980-1985	Lecturer, Pediatric Surgery Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1981-1986	Lecturer, Neurology/Pediatric Neuroradiology Lecture Series, Oklahoma Teaching Hospitals
1982-1985	Participant, Senior Radiology Resident Pre-Board Examinations, University of Oklahoma College of Medicine
1982-1986	Lecturer, Pediatric House Staff Core Lecture Series in Pediatric Radiology, Oklahoma Children's Memorial Hospital
1983-1986	Course Developer and Director, Resident Final Examination in Pediatric Radiology, University of Oklahoma College of Medicine
1985-1986	Oklahoma Diagnostic Imaging Center Lecture Series, Course Co-Developer and Co-Director
1985-1986	Oklahoma Teaching Hospitals Department of Radiological Sciences, Magnetic Resonance Imaging Lecture Series (Course Developer and Director)

1986	"Magnetic Resonance Imaging for the Referring Physician", Continuing Medical Education Seminar, Program Co-Director, Session Moderator, and Lecturer, Oklahoma Teaching Hospitals and the University of Oklahoma College of Medicine
1986-2000	Daily Neuroradiology Case Review and Consultation with Pediatric and Adolescent Medicine, Neurology, Neurosurgery, Radiology, Oncology, Radiation Therapy, Orthopedic, ORL/Head and Neck Surgery, Ophthalmology, Plastic Surgery, Oral Surgery, and Neuropathology Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA
1986-2000	Weekly Pediatric Neurology-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1986-2000	Weekly Pediatric Neurosurgery-Neuroradiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1986-2000	Weekly Pediatric Neuroncology-Neuroradiology Rounds with Pediatric Oncology, Radiation Oncology, and Neurosurgery Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital and Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA
1986-2000	Weekly Longwood Medical Area Neuroradiology Conference with Staff, Fellows, Residents, Medical Students, and visitors (The Children's Hospital, Brigham & Women's Hospital, Beth Israel Hospital, New England Deaconess Hospital, Dana-Farber Cancer Institute), Conference Co-Leader, Children's Hospital, Boston, MA
1986-2000	Monthly Pediatric ORL/Head & Neck Radiology Rounds with Staff, Fellows, Residents, Medical Students, and visitors, Conference Co-Leader, Children's Hospital, Boston, MA
1986-2000	Monthly Pediatric Radiology Difficult Case Conference (Risk Management and Quality Improvement) with Staff, Fellows, Residents, Medical Students, and visitors, Children's Hospital, Boston, MA
1986-2000	Monthly Boston Area Neuroradiology Club Case Conference with Staff, Fellows, Residents, Medical Students, and visitors (Massachusetts General Hospital)
1986-2000	Pediatric Neuroradiology Annual Lecture Series, Course Co-Director and Lecturer, for Staff, Fellows, Residents, Medical Students, and visitors.
1986-2000	Pediatric Neuroradiology Introductory Lectures for Harvard Medical Students and Rotating Radiology Residents, Radiology, Children's Hospital, Boston, MA
1986-1988	Cardiac Radiology Lecture Series, Course Developer and Lecturer, Radiology, Children's Hospital, Boston, MA
1986-1990	Magnetic Resonance Imaging Lecture Series, Course Developer, Director, and Lecturer, Radiology, Children's Hospital, Boston, MA

2000	Basic Technical and Biological Principles of Magnetic Resonance Imaging Lecture Series, Department of Radiology, Beth Israel Deaconess Medical Center, Boston, MA
2000	Pediatric Neuroradiology Resident Pre-Board Review, Department of Radiology, Beth Israel Deaconess Medical Center, Boston, MA
2000	Pediatric Neuroradiology Lectures, Neuroradiology Lecture Series, Stanford University Medical Center, Palo Alto, CA.
2001	Monthly Pediatric Neuroradiology Lecture Series for Neurology Residents & Fellows, Stanford University Medical Center, Palo Alto, CA.
2001	Pediatric Neuroradiology Lecture Series for Neurosurgery Residents and Fellows Stanford University Medical Center, Palo Alto, CA.
2001	Pediatic Head & Neck Imaging Lecture Series for ORL/Head & Neck Residents and Fellows, Stanford University Medical Center, Palo Alto, CA.
2001	Pediatric Neuroradiology Lectures, Pediatric Radiology Lecture Series, Department of Radiology, Stanford University Medical Center, Palo Alto, CA.

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8. Barnes P, Carson J, Tunell W, Smith E, Pollay M, Reynolds A, Sullivan J, Bodensteiner J, Barnes W. Occult Myelodysplasia in Children with Caudal Endodermal Syndromes". Presented at the 22nd Annual Meeting of the American Society of Neuroradiology, Boston, MA., June 1984 (American Journal of Neuroradiology 1984; 5: 673).
9. Barnes P, Lester P, Yamanashi W. "Magnetic Resonance Imaging of Posterior Fossa Masses in Children". Presented at the 70th Scientific Assembly and Annual Meeting of the Radiologic Society of North America, Washington D.C., November, 1984 (Radiology 1984; 153: 117).
10. Lester P, Barnes P, Wheatley K, Yamanashi W., Woosley R. "Intracranial Mass Lesions of Children via MRI at 0.27T". Presented at the Fourth Annual Meeting of the Society of Magnetic Resonance Imaging, San Diego, March, 1985. (Magnetic Resonance 1986; 4:41-49).
11. Barnes P, Lester P, Galloway D, Prince J, Yamanashi W. "MRI in the Management of Brainstem Neoplasia of Childhood". Presented at the 24th Annual Meeting, American Society of Neuroradiology, San Diego, California, January 1986 (American Journal of Neuroradiology 1986; 7: 542).
12. Prince J, Wegner K, Barnes P. "Contrasting Site Planning Philosophies for High-Field Strength MRI Installation". Presented at Southwestern Chapter Society of Nuclear Medicine Annual Meeting, Dallas, Texas, March 1986 (Journal of Nuclear Medicine 1986; 27: 314).
13. Barnes P, Lester P, Prince J, Galloway S, Yamanashi W. "MRI of the Spinal Neuraxis in Childhood". Presented at the Annual Meeting, Society for Pediatric Radiology, Washington, D.C., April 1986 (American Journal of Radiology 1986; 147: 871).
14. Tunell W, Barnes P, Austin J, Reynolds A. "Neuroradiologic Evaluation of Sacral Abnormalities in Imperforate Anus Complex". Presented at the Annual Meeting, American Pediatric Surgical Association, Toronto, Canada, May, 1986 (Journal of Pediatric Surgery 1986; 22: 58-61).

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15. Barnes P, Prince J, Galloway D, Ross-Duggan J, Lester P, Yamanashi W. "MR Imaging of the Pediatric Central Nervous System-Utilization Review". Scientific Presentation, Radiological Society of North America, 72nd Scientific Assembly and Annual Meeting, Chicago, Illinois, November, 1986 (Radiology 1986; 161(p):292).
16. Barnes P, Prince J, and Martel C. "High-Field MR Imaging of the Pediatric Central Nervous System". Scientific Exhibit, Radiological Society of North America 72nd Scientific Assembly and Annual Meeting, November, 1986 (Radiology 1986; 161(p): 408).
17. Barnes P, Prince J, Wilson D, Galloway D, Lester P. "The Complimentary Roles of MR and CT in Pediatric Cranio-Spinal Imaging". Presented at the Inaugural Conjoint Meeting (S.P.R.-E.S.P.R.), International Pediatric

Radiology '87, Toronto, Canada, June 1987 (Pediatric Radiology 1987; 7(#4): 345-346).

18. Hamza M, Noorani R, Bodensteiner J, Barnes P. "Benign Subdural Collection: A Cause of Macrocrania in Infancy". Presented at the 39th Annual Meeting of the American Academy of Neurology, New York, April 9, 1987 (Neurology 1987; 37: 347).

19. Noorani P, Bodensteiner J, Barnes P. "Colpocephaly: Frequency and Associated Findings". Presented (poster) at the 15th Annual Meeting of the Child Neurology Society, October 11, 1986, New Orleans. (Journal of Child Neurology 1988; 3: 100-104).

20. Bartynski W, Barnes P, Wallman J. "Cranial Computed Tomographic Findings in Autosomal Recessive Osteopetrosis". Poster presentation at the 25th Annual Meeting of the American Society of Neuroradiology, May 1987, New York. Am. J. Neuroradiology 1989; 10:543-550).

21. Hoffer F, Barnes P. "Motion-artifact Reduction at High-field Strength MRI in Children". Presented at the 74th Scientific Assembly and Annual Meeting Radiological Society of North America, Chicago, IL., November, 1988 (Radiology 1988;169(P):33).

22. Ahn S, Mantello M, Jones K, Mulkern R, Melki P, Higuchi N, Barnes P. Rapid MR Imaging of the Pediatric Brain Using Partial RF Echo Planar (PREP) Techniques. Presented at the 29th Annual Meeting, American Society of Neuroradiology, June 9, 1991, Washington, D.C. (Am. J. Neuroradiology 1992;13:1169-1178).

23. Tice H, Ahn S, Goumnerova L, Barnes P. Clinical and imaging aspects of pediatric and adolescent oligodendrogliomas. Poster presentation at the 35th Annual Meeting, Society for Pediatric Neuroradiology, June 3-4, 1992, St. Louis, MO.

24. Tice H, Jones K, Mulkern R, Schwartz R, Kalina P, Ahn S, Barnes P, Jolesz F. Evaluation of intracranial neoplasms with fast spin-echo and conventional dual spin-echo images. Presented at the 30th Annual Meeting, American Society of Neuroradiology, June 4, 1992, St. Louis, MO.

25. Kinney H, Panigrahy A, Goode R, Barnes P, Dikkes P, Korein J. Neuropathologic findings in a patient with persistent vegetative state. Poster presentation at the Annual Meeting of the American Association of Neuropathologists, June 17, 1992, St. Louis, MO. Honorable Mention, Moore Award (Best paper in clinicopathologic correlation). (J Neuropathol Exp Neurol 1992;51:345).

26. Barnes P, Dunbar S, Young Poussaint T, Kooy H, Van Herk M, Mulkern R, Loeffler J, Tarbell N. Image fusion in planning of stereotactic radiation therapy for childhood intracranial neoplasia. Presented at The Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 13, 1993, and at the American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C. Canada, May 19, 1993.

27. Jaramillo D, Barnes P, Appignani B, Young Poussaint T. Spinal dysraphism in cloacal malformation, imperforate anus, and cloacal exstrophy. Presented at The Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 13, 1993, and at the American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 18, 1993.

28. Barnes P, Tarbell N, Dunbar S, Young Poussaint T. MR imaging in treatment planning for craniospinal irradiation of childhood CNS neoplasia. Presented at The Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 14, 1993, and at the American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 19, 1993.

29. Barnes P, Appignani B, Landy H, Young Poussaint T. MR imaging in unexplained central diabetes insipidus of childhood. Presented at The Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 14, 1993, and at the American Society of Neuroradiology, 31st Annual Meeting (Idiopathic central diabetes insipidus of childhood: MR imaging), Vancouver, B.C., Canada, May 19, 1993.

30. Barnes P, Tice H, Goumnerova L. Pure oligodendrogliomas of childhood. Alternate short paper at The Society for Pediatric Radiology, 36th Annual Meeting, Seattle, Washington, May 15, 1993.

31. Tice H, Barnes P, Boyer R, Osborn A. MRI of the CNS in pediatric patients with systemic lupus erythematosis. Presented at the American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 18, 1993.

32. Barnes P, Strand R, Young Poussaint T, Estroff J. The Dandy-Walker-Blake continuum: a unified approach to retrocerebellar cystic anomalies. Presented at the American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 19, 1993.

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33. Tice H, Mulkern R, Meng J, Oshio K, Shapiro A, Barnes P, Jolesz F. Spectroscopic studies of the pituitary fossa with an inner volume spectroscopic imaging technique. Presented at the American Society of Neuroradiology, 31st Annual Meeting, Vancouver, B.C., Canada, May 20, 1993.

34. Barnes PD, Suojanen JN, Estroff J, Young Poussaint T, Burrows PE. Congenital cerebral clefts. Presented at the Society for Pediatric Radiology, 37th Annual Meeting, Colorado Springs, Colorado, April 28-May 1, 1994.

35. Estroff JA, Parad RB, Benacerraf BR, Barnes PD. Prenatal sonography of callosal dysgenesis with associated supratentorial cysts. Presented at the

Society for Pediatric Radiology, 37th Annual Meeting, Colorado Springs, Colorado, April 28-May 1, 1994.

36. Young Poussaint T, Barnes PD, Siffert JO, Pomeroy SL, Burrows PE. Outcome in delayed intracranial hemorrhage following cranial radiation therapy in children. Presented at the Society for Pediatric Radiology, 37th Annual Meeting, Colorado Springs, Colorado, April 28-May 1, 1994, and at the American Society of Neuroradiology, Nashville, Tennessee, May 3-7, 1994.

37. Treves ST, O'Tuama LA, Barnes PD, Bjornson B, Mitchell KD, Habboush I. Pediatric brain MRI/SPECT, SPECT/SPECT image fusion. Paper presented at the Society for Pediatric Radiology, 37th Annual Meeting, Colorado Springs, Colorado, April 28-May 1, 1994, and at the American Society of Neuroradiology, Nashville, Tennessee, May 3-7, 1994.

38. Barnes PD, Young Poussaint T, Burrows PE, Scott RM. Symptomatic Chiari I malformation of childhood. Paper presented at the American Society of Neuroradiology, Nashville, Tennessee, May 3-7, 1994.

39. Barnes PD, Suojanen JN, Estroff J, Young Poussaint T, Burrows PE. Congenital cerebral clefts. Poster presented at the American Society of Neuroradiology, Nashville, Tennessee, May 3-7, 1994.

40. Barnes PD, Chung T, Hoffer FA, Burrows PE, Young Poussaint T, Ohlms L. MR imaging of hemangiomas of the head and neck in childhood. Poster presented at the American Society of Neuroradiology, Nashville, Tennessee, May 3-7, 1994.

41. Tzika AA, Robertson R, Barnes PD, Burrows PE, Scott RM. Childhood moyamoya disease: hemodynamic MR imaging. Paper presented at the American Society of Neuroradiology, 33rd Annual Meeting, Chicago, Illinois, April 23, 1995 and The Society for Pediatric Radiology, 38th Annual Meeting, Washington, D.C., April 29, 1995.

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42. Robson CD, Barnes PD, Burrows PE, Hoffer FA, Paltiel HJ, Young Poussaint T, Robertson RL. MR imaging of vascular anomalies of the head and neck in childhood. Paper presented at the American Society of Neuroradiology, 33rd Annual Meeting, Chicago, Illinois, April 23, 1995 and The Society for Pediatric Radiology, 38th Annual Meeting, Washington, D.C., April 29, 1995.

43. Tzika AA, Barnes PD, Tarbell NJ, Nelson SJ, Scott RM. Multivoxel Proton Spectroscopy of childhood brain tumors. Derek Harwood-Nash Award for Outstanding Pediatric Neuroradiology Paper. Paper presented at the American Society of Neuroradiology, 33rd Annual Meeting, Chicago, Illinois, April 24, 1995.

44. Young Poussaint T, Barnes PD, Robertson RL, Robson CD, Walters G. Hemorrhagic pituitary adenomas of adolescence. Paper presented at the American Society Neuroradiology, 33rd Annual Meeting, Chicago, Illinois, April 24, 1995.

45. Huppi PS, Tsuji MK, Kapus T, Barnes P, Zientara G, Kikinis R, Jolesz F, Volpe JJ. 3D-MRI, a new measure of brain development in newborns. Paper presented at the Society of Pediatric Research, 64th Annual Meeting, San Diego, CA, May 1995.

46. Huppi PS, Tsuji MK, Kapur T, Barnes P, Jakab M, Zientara G, Kikinis R, Jolesz F. Quantification of changes in postnatal brain development in preterm infants using adaptive segmentation of MRI data. Paper presented at the Proceedings of the Third Annual Scientific Meeting of the Society of Magnetic Resonance, Nice, France, August 1995.

47. Medina LS, Barnes PD, Pinter J, Davis R, Zurakowski D. Clinical practice guidelines for imaging in children with headache. Paper presented at the Radiological Society of North America, 81st Annual Meeting, Chicago, Illinois, November 28, 1995.

48. Packard AB, Connolly LP, Bar-Sever Z, Barnes PD, Holmes G, Treves ST. Ictal and interictal Tc-99m ECD SPECT in pediatric patients with medically refractory epilepsy without focal MR imaging abnormalities. Paper presented at the Radiological Society of North America, 81st Annual Meeting, Chicago, Illinois, November 28, 1995.

49. Tzika AA, Barnes PD, Tarbell NJ, Goumnerova LC, Scott RM, Nelson SJ, et al. Spectroscopic and hemodynamic MR characterization of pediatric brain tumors. Paper presented at the Radiological Society of North America, 81st Annual Meeting, Chicago, Illinois, November 28, 1995.

50. Burrows PE, Barnes PD, Ezekowitz RA, Mulliken JB. Intracranial vascular anomalies in patients with cervicofacial hemangiomas. Paper presented at the Radiological Society of North America, 81st Annual Meeting, Chicago, Illinois, November 29, 1995.

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51. Medina LS, Pinter J, Zurakowski D, Davis RG, Barnes PD. Clinical predictors of surgical space-occupying lesions and the role of Neuroimaging in children with headache. Paper presented at the SPR/IPR '96 Meeting, Boston, MA, May 1996.

52. Robson CD, Bakardjiev AI, Barnes PD, Kim FM, Robertson RL, Poussaint TY, et al. MR imaging changes after stereotactic radiation therapy for brain tumors in children. Paper presented at the SPR/IPR '96 Meeting, Boston, MA, May 1996.

53. Robson CD, Pohl-Koppe A, Barnes PD, Thiele E, Robertson RL, Burchett S. The role of brain MR imaging in the differential diagnosis of acute viral encephalitis and acute disseminated encephalomyelitis in children.

Paper presented at the American Society of Neuroradiology, 34th Annual Meeting, Seattle, Washington, June 23, 1996.

54. Robson CD, Bakardjiev AI, Barnes PD, Kim FM, Robertson RL, Poussaint TY. MR imaging changes after stereotactic radiation therapy for brain tumors in children. Paper presented at the American Society of Neuroradiology, 34th Annual Meeting, Seattle, Washington, June 24, 1996.

55. Klufas RA, Barnes PD, Robson CD, Kim FM, Robertson RL, Poussaint TY. MR imaging of spinal cord gangliogliomas of childhood. Paper presented at the American Society of Neuroradiology, 34th Annual Meeting, Seattle, Washington, June 25, 1996.

56. Robertson RL, Burrows PE, Barnes PD, Robson CD, Scott RM. Angiographic changes following pial synangiosis in moyamoya syndrome. Poster presented at the American Society of Neuroradiology, 34th Annual Meeting, Seattle, Washington, June 1996.

57. Huppi PS, Tsuji MK, Barnes P, Kikinis R, Jolesz F, Volpe JJ. Quantitative assessment of brain development in multiple gestation babies using in vivo 3-dimensional MRI (3D-MRI). Paper presented at the European Society for Pediatric Research and the European Society for Pediatric Intensive Care, Annual Meeting, Lyon, France, September 1996.

58. Tzika AA, Vajapeyam S, Barnes PD, Tarbell NJ, Goumnerova LC, Anthony DC. Pediatric brain tumor response to treatment with proton MR spectroscopy. Paper presented at the Radiological Society of North America, 82nd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 2, 1996.

59. Kikinis R, Huppi P, Barnes PD, Volpe JJ, Jolesz FA. MR-based quantification of brain development in multiple-gestation preterm infants. Paper presented at the Radiological Society of North America, 82nd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 2, 1996.

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60. Medina LS, Mulkern RV, Strife KR, Zurakowski D, Barnes PD. Database prescan: a time-efficient alternative to brain MR imaging autoprescan. Paper presented at the Radiological Society of North America, 82nd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 2, 1996.

61. Barnewolt CE, Kim FM, Barnes PD, Taylor GA. Potential role of color Doppler sonography in defining the location of extracerebral fluid collections in infants. Paper presented at the Radiological Society of North America, 82nd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 3, 1996.

62. Medina LS, Zurakowski D, Strife KR, Robertson RR, Young Poussaint T, Barnes PD. Efficacy of fast-screening brain MR imaging in children with space-occupying lesions: blinded comparative analysis. Paper presented at the Radiological Society of North America, 82nd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 6, 1996.

63. Glasier CM, Barnes PD, Allison JW. Rathke cleft cysts in young patients: CT, MR imaging, and clinical-pathologic correlation. Paper presented at the Radiological Society of North America, 82nd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 6, 1996.

64. Huppi PS, Warfield S, Zientara GP, Taranto RJ, Barnes PD, Kikinis R, Jolesz FJ. Cortical development in early human brain development: surface and volume changes. Paper presented at the Proceeding of the Fifth Annual Scientific Meeting of the International Society for Magnetic Resonance in Medicine, Vancouver, B.C., Canada, April 12-18, 1997.

65. Alberico RA, Barnes PD, Robertson RL, Burrows PE. Kirkpatrick Young Investigator Award. Dynamic cerebrovascular imaging in pediatric patients with use of helical CT angiography. Paper presented at the Society for Pediatric Radiology, 40th Annual Meeting, St. Louis, Missouri, May 15, 1997.

66. Robson CD, Weber AL, Robertson RL, Barnes PD. The radiologic evaluation of parotid masses in children. Paper presented at the American Society of Neuroradiology/American Society of Head and Neck Radiology, 35th Annual Meeting, Toronto, Ontario, Canada, May 18, 1997.

67. Alberico RA, Barnes PD, Robertson RL, Burrows PE. Dynamic cerebrovascular imaging in pediatric patients with use of helical CT angiography. Paper presented at the American Society of Neuroradiology, 35th Annual Meeting, Toronto, Ontario, Canada., May, 1997.

68. Robertson RL Chavali R, Robson CD, Burrows PE, Barnes PD, Poussaint TY, Scott RM. Cerebral angiographic technique and complications in childhood Moyamoya disease. Paper presented at the American Society of Neuroradiology, 35th Annual Meeting, Toronto, Ontario, Canada, May 19, 1997.

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69. Carrico JB, Burrows PE, Mulliken JB, Robertson RL, Barnes PD. Intracranial vascular anomalies in patients with orbital lymphatic malformation. Poster presentation at the American Society of Neuroradiology, 35th Annual Meeting, Toronto, Ontario, Canada, May 21, 1997.

70. Levine D, Sher SI, Semeika RC, Li W, Edelman RR, Barnes PD. Normal fetal neuroanatomy with ultrafast fetal MR imaging with HASTE. Scientific exhibit presentation at the Radiological Society of North America, 83rd Scientific Assembly and Annual Meeting, Chicago, Illinois, November 30-December 5, 1997.

71. Tzika AA, Vajapeyam S, Barnes PD, Scott RM, Goumnerova LC, Tarbell NJ. Anatomic, metabolic and hemodynamic evaluation of childhood brain neoplasms during therapy. Paper presented at the Radiological Society of North America, 83rd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 1, 1997.

72. Poussaint TY, Kowal JR, Barnes PD, Zurakowski D, Anthony DC, Goumnerova LC. Tectal tumors of childhood: clinical and imaging followup. Paper presented at the Radiological Society of North America, 83rd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 1, 1997.

73. Levine D, Barnes PD, Madsen JR, Hulka CA, Li W, Edelman RR. HASTE MR imaging improves sonographic diagnosis of fetal central nervous system anomalies. Scientific Exhibit, Cum Laude Citation, and paper presented at the Radiological Society of North America, 83rd Scientific Assembly and Annual Meeting, Chicago, Illinois, December 2, 1997.

74. Medina LS, Al-Orfali M, Zurakowski D, Poussaint TY, DiCanzio J, Barnes PD. MR imaging standards for children and young adults with suspected occult dysraphic myelodysplasias. Paper presented at The Society for Pediatric Radiology, 41st Annual Meeting, Tucson, Arizona, May 7-9, 1998 and the American Society of Neuroradiology, 36th Annual Meeting, Philadelphia, Pennsylvania, May 17-21, 1998.

75. Levine D, Barnes P. Cortical development and maturation in normal and abnormal fetuses as assessed with prenatal MR imaging. Poster presentation at the American Society of Neuroradiology, 36th Annual Meeting and Symposium Neuroradiologicum XVI, Philadelphia, Pennsylvania, May 15-21, 1998.

76. Levine D, Barnes P, Hulka C, Madsen J, Edelman R. Evaluation of fetal central nervous system abnormalities with ultrafast MRI. Poster presentation at the American Society of Neuroradiology, 36th Annual Meeting and Symposium Neuroradiologicum XVI, Philadelphia, Pennsylvania, May 15-21, 1998.

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77. Robertson RL, Maier SE, Mulkern RV, Robson CD, Barnes PD. Line scan spin-echo diffusion imaging of the brain in children. Paper presented at the Society for Pediatric Radiology, 41st Annual Meeting, Tucson, Arizona, May 7-9, 1998 and the American Society of Neuroradiology, 36thAnnual Meeting and Symposium Neuroradiologicum XVI, Philadelphia, Pennsylvania, May 15-21, 1998.

78. Robson CD, Robertson RL, Hazra R, Reid J, Barnes PD, Jones DT, Husson R. The radiological evaluation of nontuberculous mycobacterial infection of the head and neck in immunocompetent children. Paper presented at the American Society of Head and Neck Radiology, Annual

Meeting, Phoenix, Arizona, April 1-5, 1998 and the American Society of Neuroradiology, 36th Annual Meeting, Philadelphia, Pennsylvania, May 17-21, 1998.

79. Robson CD, Reid J, Robertson RL, Barnes PD, Ferraro N. The radiologic evaluation of chronic sclerosing osteomyelitis of the mandible in children. Paper presented at the American Society of Head and Neck Radiology, Annual Meeting, Phoenix, Arizona, April 1-5, 1998 and the American Society of Neuroradiology, 36th Annual Meeting, Philadelphia, Pennsylvania, May 17-21, 1998.

80. Poussaint TY, Yousef N, Barnes PD, Scott RM, Tarbell NJ. Cervicomedullary astrocytomas of childhood: clinical and imaging follow-up. Paper presented at the American Society of Neuroradiology, 36th Annual Meeting, Philadelphia, Pennsylvania, May 17-21, 1998.

81. Levine D, Abbott J, Barnes P, Mehta TS, Hulka DA, Wong G, et al. Ultrafast MRI of fetal CNS anomalies: In which categories of sonographic abnormalities is MRI likely to be helpful? Scientific Exhibit and Scientific Paper presented at the Radiological Society of North America, Chicago, IL, Nov. 1998.

82. Levine D, Abbott J, Barnes PD, Mehta TS, Hulka CA, Edelman RR, et al. New uses of fast MRI in obstetric diagnosis, Scientific Exhibit presented at the Radiological Society of North America, Chicago IL, Nov. 1998.

83. Robertson RL, Ben-Sira L, Schlaug G, Robson CD, Maier SE, Mulkern RV, Barnes PD. Diffusion imaging in neonates with suspected hypoxic-ischemic brain injury. Paper presented at The Society for Pediatric Radiology, 42nd Annual Meeting, Vancouver, B.C., Canada, May 16, 1999.

84. Ben-Sira L, Robertson RL, Mulkern RV, Maier SE, Barnes PD. Diffusion imaging in new-onset childhood seizures. Paper presented at The Society for Pediatric Radiology, 42nd Annual Meeting, Vancouver, B.C., Canada, May 16, 1999.

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85. Barnes PD, Tzika AA, Robertson RL, Poussaint TY, Robson CD, Goumnerova LC, Scott RM. Relationship of MR imaging and proton MR spectroscopy in the presurgical evaluation of neuroepithelial tumors of childhood. Paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA, May 23, 1999.

86. Tzika AA, Poussaint TY, Zurakowski D, Goumnerova LC, Tarbell NJ, Scott RM, Black P.MCL, Barnes PD. Assessment and prediction of pediatric brain neoplasm therapeutic response using proton MR spectroscopic imaging. Paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA, May 23, 1999.

87. Robson CD, Mulliken JB, Robertson RL, Proctor MR, Barnes PD. Prominent emissary veins in Crouzon Syndrome. Paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA, May 23, 1999.

88. Robertson RL, Ben-Sira L, Schlaug G, Maier SE, Mulkern RV, Duplessis A, Barnes PD, Robson CD. Line scan diffusion imaging of the brain in neonatal cerebral infarction. Derek Harwood-Nash Award for Outstanding Pediatric Neuroradiology Paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA, May 24, 1999.

89. Tzika AA, Robertson FL, Burrows PE, Barnes PD, Scott RM. Multilevel brain perfusion-weighted imaging in children with Moyamoya disease after pial synangiosis. Paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA, May 24, 1999.

90. Tzika AA, Robertson RL, Ben-Sira L, Poussaint TY, Robson CD, Barnes PD. Proton MR spectroscopy on neonates with suspected cerebral ischemic encephalopathy. Paper presented at the ASNR/ASPNR Annual Meeting, San Diego, CA, May 24, 1999.

91. Zientara GP, Murphy BP, Maier SE, Huppi PS, Barnes PD, Volpe JJ, Jolesz FA. Diffusion tensor MRI of the human cervical spinal cord in vivo in preterm newborns. Poster presentation at the International Society for Magnetic Resonance in Medicine, 7th Scientific Meeting and Exhibition, Philadelphia, PA, May 22-28, 1999.

92. Murphy BP, Zientara GP, Huppi PS, Maier SE, Barnes PD, Jolesz FA, Volpe JJ. Diffusion weighted MRI to assess cerebral white matter injury in very low birth weight infants. Poster presentation at the International Society of Magnetic Resonance in Medicine, 7th Scientific Meeting and Exhibition, Philadelphia, PA, May 22-28, 1999.

93. Hong H-S, Mulkern RV, Ma JF, Robertson RL, Robson CD, Barnes PD. Phase sensitive inversion recovery magnetic resonance imaging of the pediatric brain. Poster presentation at the International Society of Magnetic Resonance in Medicine, 7th Scientific Meeting and Exhibition, Philadelphia, PA, May 22-28, 1999.

94. Tzika AA, Petridou N, Robertson RL, Duplessis A, Poussaint TY, Robson CD, Barnes PD. Proton MRS in neonates with suspected cerebral ischemic encephalopathy. Poster presentation at the International Society of Magnetic Resonance in Medicine, 7th Scientific Meeting and Exhibition, Philadelphia, PA, May 22-28, 1999.

95. Tzika AA, Vajapeyam S, Zurakowski D, Poussaint TY, Goumnerova L, Barnes PD, Anthony DC, Billett AL, Tarbell NJ, Scott RM, Black P. McL. Predictors of tumor growth as assessed by proton MRS in pediatric brain tumors. Poster presentation at the International Society of Magnetic

Resonance in Medicine, 7th Scientific Meeting and Exhibition, Philadelphia, PA, May 22-28, 1999.

96. Vajapeyam S, Mulkern RV, Robertson RL, Barnes PD, Rivkin MJ. Effect of signal fluctuations from the eyes on fMRI data and post-processing. Poster presentation at the International Society of Magnetic Resonance in Medicine, 7th Scientific Meeting and Exhibition, Philadelphia, PA, May 22-28, 1999.

97. Panigrahy A, Back SA, Barnes PD, Robertson RL, Sleeper S, Volpe J. Volumetric comparison of periventricular MR T2 / Flair signal hyperintensities between age matched term and premature infants. Paper presentation at the Radiologic Society of North America annual meeting, Chicago IL, Dec. 1999.

98. Rybicki FJ, Mulkern RV, Robertson RL, Robson CD, Barnes PD. T2-weighted fast three-point dixon MR imaging of the retrobulbar space: comparison with fast spin echo inversion recovery. Paper presentation at the Radiologic Society of North America annual meeting, Chicago IL, Dec. 1999.

99. Robertson, RL, Maier SE, Mulkern RV, Robson CD, Vajapayem S, Barnes PD. Prominent emissary foramina in syndromic craniosynostosis: correlation with phenotypic and molecular diagnosis. Paper presentation at the American Society of Neuroradiology, Atlanta, GA, April 2000, and at the Joint International Conference and Symposium of the American Society of Head and Neck Radiology and the European Society of Head and Neck Radiology, Washington DC, May 2000.

100. Robertson RL, Maier SE, Mulkern RV, Robson CD, Vajapayem, Barnes PD. Line scan diffusion imaging of the spine in children. Paper presentation at the American Society of Neuroradiology annual meeting, Atlanta GA, April 2000.

101. Tzika AA, Poussaint TY, Robertson RL, Barnes PD. Correlatiion between Gd-DTPA enhancement an other MRI / MRS derived parameters in the assessment of pediatric brain tumors. Paper presentation at the American Society of Neuroradiology annual meeting, Atlanta GA, April 2000.

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102. Tzika AA, Cheng LL, Poussaint TY, Robertson RL, Barnes PD, Gonzalez RG. Comparison of in vivo proton MRS of pediatric brain tumors with ex vivo MRS of intact biopsy tumor samples. Paper presentation at the American Society of Neuroradiology annual meeting, Atlanta GA, April 2000.

103. Levine D, Mehta TS, Trop K, Li W, Abbott J, Barnes PD. Fast MRI of fetal CNS anomalies: results of 149 fetal examination. Poster presentation at the Radiologic Society of North America annual meeting, Chicago, IL, Nov. 27, 2000.

104. Trop I, Levine D, Mehta TS, Barnes PD. Sonographic and MR evaluation Of the fetal ventricle: it's more than just a measurement. Poster Presentation at the Radiologic Society of North America annual meeting, Chicago, IL, Nov. 27, 2000.

105. Panigrahy A, Barnes PD, Robertson RL, Sayre JW, Back SA, Volpe JJ. Volumetric MR correlates of neuromotor abnormalities in children with Periventricular white matter T2 hyperintensities. Paper presentation at the Radiologic Society of North America annual meeting, Chicago, IL, Nov. 29, 2000.

106. Panigrahy A, Barnes PD, Robertson RL, Sayre JW. Differential MR characteristics associated with periventricular T2 hyperintensities in children with spastic diplegia. Paper presented at the 39th annual ASNR meeting, ASPNR pediatric scientific session, April 23, 2001.

107. Panigrahy A, Barnes PD, Robertson RL, Sayre JW. Comparative, quantitative MR analysis of the corpus callosum in children with spastic diplegia: a correlation with cerebral white matter volume. Poster presentation at the Radiologic Society of North America, 87th Scientific Assembly and Scientific Meeting, Chicago, IL, Dec. 27, 2001.

108. Barnes P, Arzoumanian Y, Woolley K, Mirmiran M, Atlas S, Moseley M, Ariagno R. MRI (DTI) in preterm infants may predict later cerebral palsy. Paper presentation, Society for Pediatric Radiology 45th Annual Meeting, Philadelphia PA, May 29, 2002.

109. Barnes P, Ment L, Grant E, Slovis T, Bada H, Papile A, et al. Neuroimaging of the neonate: an evidence-based practice parameter. Paper presentation, Society for Pediatric Radiology 45th Annual Meeting, Philadelphia PA, May 29, 2002.

110. Barnes P, Dermon J, Spielman D. Spatiotemporal mapping of cerebral maturation in childhood using 2D MR spectroscopic imaging – preliminary report. Scientific exhibit, Society for Pediatric Radiology 45th Annual Meeting, Philadelphia PA, May 2002.

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41. Barnes PD. Congenital and Developmental Conditions of the Spine and Spinal Cord. Concepts in Neuroradiology, Head & Neck Radiology, and Clinical Functional MRI and Spectroscopy. The Massachusetts General Hospital and Harvard Medical School Post-Graduate Course Syllabus, Boston, MA, September 16, 1998.
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45. Barnes PD. Potential Pitfalls in Pediatric Neuroradiology, MRI/CT Update Post-graduate Course Syllabus, Brigham & Women's Hospital, Harvard Medical School, Boston, MA, October 29, 1999.

46. Barnes PD. Neuroimaging and the timing of fetal brain injury, & The Neuroimaging expert, Birth Injury and the Law VIII Course Syllabus, Medical Intelligence Corporation Conference, Las Vegas, Nevada October 19, 2000.
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50. Barnes PD. MDCT applications in Pediatric Neuroradiology (Brain, Spine, Head & Neck). 6th Annual International Symposium on Multidetector-Row CT. Stanford University Medical Center Course Syllabus, San Francisco CA, June 23, 2004.
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53. Barnes P.Child abuse: the role of neuroimaging in the clinical and forensic evaluation of suspected nonaccidental injury including its mimics. 13th Annual Pediatric Update, Lucile Packard Children's Hospital and Stanford University Medical Center Course Syllabus, July 8, 2005.
54. Barnes P. Imaging of the Pediatric Central Nervous System and Head & Neck: MRI, CT, US, Nuclear Medicine – Which to do? 14th Annual Pediatric Update, Lucile Packard Children's Hospital and Stanford University Medical Center Course Syllabus, July 21 , 2006.
55. Barnes PD. Lecturer. Advances in Pediatric CT and MRI: Head & Neck Imaging I (Orbit, Sinus, Ear), Head & Neck Imaging II (Face & Neck), Spine Imaging I (Developmental Anomalies), Spine Imaging II (Acquired Conditions), Brain Imaging III (Acute neurologic conditions – Trauma [including child abuse], hemorrhage, vascular disease), Brain Imaging V (Subacute neurologic conditions – Tumors, epilepsy). Department of Radiology, Stanford School of Medicine Postgraduate Course. Las Vegas, Nevada, March 17, 2007. Course Syllabus.

EXHIBIT S

Mary Had a Little Lamb
Mary had a little lamb,
Little lamb, little lamb,
Mary had a little lamb,
Its fleece was white as snow.
Everywhere that Mary went,
Mary went, Mary went,
Everywhere that Mary went,
The lamb was sure to go.

Shapes

VIRGINIA:

IN THE CIRCUIT COURT OF FAIRFAX COUNTY,

TRUDY MUNOZ RUEDA

Petitioner,

At Law No. 2012-17074

HAROLD W. CLARKE
DIRECTOR OF VIRGINIA DEPT OF CORRECTIONS

Respondent.

AFFIDAVIT

1. My name is Erin Whitmer. Trudy Munoz Rueda was convicted of charges of child abuse and child cruelty against my infant son, Noah Whitmer.

2. Noah was between four and five months old (20 weeks and 2 days) when he was injured. Prior to Noah's hospitalization on April 20, 2009, he appeared healthy and had never even had a cold. He had no fever in the days preceding his injuries, and was not unusually fussy or irritable as suggested in the habeas petition. He had no fever and was happy and alert

EXHIBIT A

when I left him at Ms. Rueda's on the morning of April 20, 2009. He currently has no diagnosis of any bleeding disorders or genetic problems.

3. I saw no indication that Noah had a respiratory infection or any other illness on or in the days before April 20, 2009. We had noticed that Noah would sometimes sound as though he still had fluid in his lungs or that he was "wheezing," which we had mentioned during his 4-month-check-up. It was only after dealing with this issue for several months, up until he was about 9 months old (five months after his hospitalization for his injuries), and after speaking to a speech therapist, that we suspected this "wheezing" was due to the fast-flowing Dr. Brown's bottle nipples. Once we switched to Avent slower flowing nipples in September of 2009 Noah never had this issue again, despite drinking from bottles until he was over two years old.

4. Ms. Rueda, his child care provider at the time he was injured, agreed to update us as to any problems she noticed with Noah while he was in her care. She typically provided us with notes showing what he had eaten, when and for how long he had napped, and what his mood was on that particular day. In the days before April 20, 2009 she never said that Noah was sick or unusually fussy; nor did she send home any notes indicating any such problems. In fact, her notes always indicated that Noah had been "Happy."

5. Despite the suggestion in the petition that Noah had stopped eating prior to his injury, Noah was a voracious eater, even in the days before his injury. While it is true that Noah's interest in formula and bottles had waned since beginning solid foods a couple weeks before, cutting his fluid consumption down quite a bit, Noah was eating generous meals of oatmeal, peas, butternut squash, or avocado. Prior to being dropped off at daycare on the 20th, his interest in formula had returned, as he drank the typical 8-ounce bottle of formula that morning.

AFFIANT

Erin M Whitmer

Erin Whitmer

Subscribed and sworn to before me, a Notary Public in the County/City of Henrico in the State of Virginia, on this 14 day of January, 2013, by Erin Whitmer.

WILLIAM REED LESTER
NOTARY PUBLIC
REG. #7510281
MY COMMISSION EXPIRES
11/30/15
COMMONWEALTH OF VIRGINIA

Notary Public

My commission expires:
11/30/15

Response to Petition For a Writ of Habeas Corpus
Date: January 25, 2013
Case: Trudy Munoz Rueda v Harold W Clarke
(Whitmer, Noah)

ZÉDA MEDICAL INSTITUTE
William E. Hauda II, MD

RESPONSE

Shaken Baby Syndrome is a well-described entity, based upon extensive medical literature and scientific study, which is a part of a broader category of non-accidental or abusive head trauma. Literally hundreds of scientific articles, medical textbooks, and medical society policies have been published regarding its diagnosis and treatment. There will fortunately never be a randomized controlled clinical trial to determine the mechanisms of injury and level of force required to cause brain injury by shaking an infant. The Petition for Writ of Habeas Corpus states that it is a "controversial scientific hypothesis that has yet to be validated," as though the author is expecting a randomized controlled clinical trial of shaking infants to satisfy a supposed lack of validation. Recent studies involving infants with traumatic brain injuries have documented multiple care-providers stating that they "violently shook" an infant.[1] Hundreds of cases where a care giver describes or confesses to violently shaking a child have been described in the literature. A case of shaking an adult has also been described. A common finding in many of these cases is the presence of subdural hemorrhages, retinal hemorrhages, and brain injury with a sudden onset of symptoms without any other reasonable diagnostic explanation for the presences of all of the findings.

The use of evidence based medicine requires "*the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence based medicine means integrating individual clinical expertise with the best available external clinical evidence from systematic research.*"[2] The petition cites articles that misrepresent the scientific understanding of abusive head injury from shaking. The petition also fails to include additional injuries present when Noah was evaluated by only referring to the "triad" of findings. As discussed below, a deeper brain injury and cortical contusions are also frequently present in repetitive rotational head trauma, and these were found in Noah. While there are other diagnostic possibilities

EXHIBIT 3

for retinal hemorrhages, subdural hemorrhages, and brain injuries in isolation or in conjunction, clinicians must evaluate the patient in the context of the available clinical history, additional clinical findings or lack of clinical findings, and results of diagnostic evaluation. Clinician must base their evaluation on the relative probabilities of disease, not on the possibility or rarity of disease.

The petition states "it is a supposition of how these injuries might occur that is now known to be at odds with biomechanical science, pediatric neurology, and ophthalmology." This statement is simply untrue. While discussion exists in the medical literature about features and modeling of repetitive rotational head trauma, multiple biomechanical models are demonstrating the types of injuries seen in abusive head trauma including models based upon two dimensional finite element models and systems models. Even animal models using different species has demonstrated the occurrence of injuries under non-impact shaking forces. Confining the discussion to "pediatric neurology" seems to discount the numerous articles published in pediatric critical care, pediatric trauma, pediatric neurosurgery, pediatric emergency medicine, and pediatrics. All of these disciplines have peer reviewed articles discussing the clinical features and findings in abusive head trauma including injury by shaking. Regarding ophthalmology, many peer reviewed articles have been published documenting the increased frequency of severe retinal hemorrhages in children who have traumatic brain injury but who have not been injured by a witnessed or known episode of trauma such as motor vehicle crashes, crush injuries, or a fall from greater than 1 story.

The petition further states that "while almost no one in the scientific mainstream questioned SBS's existence and reliability in 2000, today questioning SBS is in the mainstream." [page 5] In support of this statement three media sources, four law sources, and six medical articles are cited. [pages 5 and 6] The petition fails to mention that during those same years, hundreds of articles have been published on Shaken Baby Syndrome,

the vast majority supporting its relevance in the diagnostic consideration of a child with intracranial hemorrhage. Textbooks continue to publish chapters discussing Shaken Baby Syndrome, although they may also use a more inclusive term such as non-accidental head injury or abusive head injury. The petition states "ongoing medical research in the last decade has disproven virtually every hypothesis in support of the existence of Shaken Baby Syndrome." [page 7] This statement is erroneous, misleading, and untrue. In fact, multiple articles have been published supporting the care-giver's description of sudden limpness after shaking, the high frequency of severe retinal hemorrhages in cases of suspected shaking injury in contrast to the low frequency in other conditions such as non-traumatic intracranial hemorrhage and witnessed accidental trauma, animal modeling of rotational head injury showing brain and eye trauma without impact, and biomechanical modeling of brain injury caused by repetitive harmonic movements of the brain within the skull. Far from being "disproven" the forces and mechanisms involved in subdural hemorrhage formation, retinal hemorrhage formation, apnea, traumatic axonal injury, and brain swelling are becoming clearer and better understood.

The petition discusses Noah's illness prior to being admitted to the hospital as though it was the cause for his findings. The petition does not discuss how this may have occurred other than the presumption that if Noah had an infection it may have caused or contributed to his cortical vein thrombosis. As stated in the petition "earlier accidental trauma can provide a pre-existing condition increasing the chances that something (such as an infection or dehydration) will trigger a venous thrombosis." [page 21] Under this presumption, (1) a pre-existing injury must be present, (2) an intercurrent illness must occur which (3) causes the venous thrombosis to occur leading to (4) subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, and injury to the corpus callosum.

As to (1), no evidence was seen to implicate a pre-existing condition caused by accidental trauma. Noah did not have a pre-existing head trauma as claimed in the petition. [pages 20 and 21] Noah had no evidence of any old trauma to his brain when evaluated by MRI or CT scan.

Supposition (2) requires "something" which will trigger the venous thrombosis. While an infection can cause venous thrombosis, the mechanism is by local inflammation in the region of the vein which causes an inflammatory and coagulation response to the inflamed vein. For cortical veins, the infectious conditions would not include pneumonia. An infection elsewhere in the body, in Noah's case a possible respiratory infection would not be a medically plausible cause for a cerebral vein thrombosis. Noah did not have meningitis. While a lumbar puncture was never performed, Noah recovered after a few days of antibiotics. The treatment for bacterial meningitis is weeks not days. If Noah had meningitis, as suggested by the petition, he would have become sicker when the antibiotics were stopped after a few days, not better. Both staphylococcal meningitis and pneumococcal meningitis require prolonged courses of antibiotics.[3] Noah did not have bacteremia (bacteria in his blood stream) as proven by his blood cultures lacking any bacterial growth.

Dr. Barnes stated in his affidavit that he considers multiple possibilities for the pre-existing condition such as vaccination, the mother's pregnancy, labor and delivery, feeding problems, colds. "The possibility that he had some pre-existing condition could not be diagnosed unless medical personnel did a careful and through family history." [Item 17, page 6 of 8] Every child will have a medical history of pregnancy (gestation) and delivery (birth). He does not elucidate regarding the important conditions that may occur during pregnancy or delivery. Similarly, he lists vaccination as a "cause for concern" in an infant as well as feeding problems and colds. He incorrectly asserts that "when an infant is vaccinated, that is essentially given them a mild infection." [Item 17,

page 6 of 8] The vaccines recommended to be given to infants at the 4 month visit in the United States do not cause infection.[4] Only the oral rotavirus vaccine is a live virus, but the FDA and CDC specifically state with the rotavirus vaccination that it not known to cause infection in people.[5] Infants are expected to experience an immunologic reaction so that protective antibodies develop from the vaccines; the vaccines do not cause an infection from a virus or bacteria. Item 16 demonstrates that Dr. Barnes does not even know which immunizations are recommended for infants at 4 months of age as both the pneumococcal and measles vaccinations are not recommended at 4 months of age. Dr. Barnes statements about vaccinations are unsupported by published recommendations, published vaccine information, and basic immunology of how vaccines function.

Item (3) the causation of the cerebral venous thrombosis was most probably due to the same trauma that caused the subdural hemorrhage to appear. While Dr. Barnes "maintains that he has *never seen a case where shaking – however violent – caused a venous thrombosis*," [page 28] any form of trauma or injury to a vein may lead to thrombosis. Vein thrombosis is seen after penetrating trauma, such as intravenous catheter placement, and after blunt trauma, such as cerebral contusions. The basic tenet of Virchow's triad for development of an intravessel thrombus are (1) alterations to normal blood flow (stasis), (2) injuries to the vascular endothelium (vein), and (3) alterations in the consistency of the blood (coagulation). No requirement exists that all three elements must be present for thrombus to occur; the interaction in the body to promote hemostasis is complex, multifactorial, and interactive. Trauma to the vein may occur by blunt trauma, penetrating trauma, or tension (stretching). Stating that the presence of a cortical vein thrombosis therefore excludes trauma, and thereby requires a medical diagnosis is medically unsound. Any traumatic force that can cause injury to a vein can induce thrombosis within the vein. Noah had bilateral occipital cortical contusions identified on the brain MRI as well as a left parietal cortical contusion. The presence of cerebral cortical contusions indicates that some blunt force injury must have occurred to Noah's

brain, whether by external impact or by internal rotational impact against the inside of the skull. External impact does not need to occur, however. Biomechanical modeling as well as clinical experience shows that movement of the brain inside the skull can cause the brain to impact against the inside of the skull. Cases of manual shaking may also occur with blunt impact such a when a child is thrown down or away from the care-giver after the manual shaking. Impact trauma may not be associated with visible external evidence of impact.[6] Dr. Barnes states that "impact trauma is a pretty obvious cause of venous thrombosis because there tends to be bruising of some kind or skull fracture." [Item 6, page 3 of 8] Dr. Barnes statement is unsupported by published studies as well as common clinical experience that impact trauma causing skull fractures more frequently lacks visible bruising. A lesser degree of impact injury, where no skull fracture occurs, would be even less likely to be associated with visible bruising. Thus, a lack of visible external trauma does not imply that external impact trauma did not occur.

Lastly, (4) Noah's single small vein thrombosis could not have caused the subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, and corpus callosum injury that were present on his studies. The importance of the cortical vein thrombosis must be taken in context of the other findings. The vein was small and singular, and no involvement of the larger sinuses was found. When cerebral vein thrombosis causes severe symptoms, the vein that is occluded is large (such as the superior sagittal sinus or the sigmoid sinus) and large areas of the cerebral cortex become ischemic from venous stasis and hypoxic injury. Noah had no such findings on his studies. In fact, the cortical vein thrombosis was small, singular, and in a single bridging vein which would serve a small portion of the cerebrum as evidence by the small area of subjacent ischemia. Dr. Barnes states in his affidavit that "it appears to me that Noah suffered a series of strokes from venous thrombosis." [Item 5, page 3 of 8] The results of the CT and MRI do not support this contention. No radiologist found multiple areas of ischemic injury to suggest a "series of strokes." A single area of

ischemia was identified underlying the described cortical vein with suspected thrombosis. There were not multiple areas to suggest or even consider a "series" of strokes. He also suggests that a "pre-existing collection from birth" could have new hemorrhaging; [item 5, page 3 of 8] however, again, no radiologist, neurosurgeon, neurologist, or other physician described any old collection on Noah's head CT or MRI. There simply was no evidence of an old fluid collection or injury. Noah's subdural hemorrhages were acute and therefore inconsistent with a suggestion that they represented an old injury.

A study of patients with intracranial vein thrombosis found no association of vein thrombosis with subdural hemorrhages.[7] The probability remains that Noah was injured by shaking and possibly by shaking and subsequent impact. Pediatric patients, particularly those that survive the blunt impact injury, commonly do not have visible evidence of impact externally. A primary tenent of pediatric trauma is that the lack of visible external trauma does not imply that serious internal trauma is absent. Thus, impact may not result in visible external manifestations of the impact. In a series of children with known skull fractures, less than 50% had visible evidence of impact.[6] In summary, trauma is a completely plausible explanation for the finding of a single small cerebral cortical vein thrombosis. Such a small thrombosis would not cause the additional findings that Noah had. Regardless of the cause for the thrombus, Noah had other clinical findings and historical features consistent to a reasonable degree of medical certainty with severe repetitive rotational head trauma such as from shaking. To a reasonable degree of medical certainty, the cortical vein thrombosis was not the cause of the subdural hemorrhage, but the cause for the subdural hemorrhage (trauma) was the cause for the cortical vein thrombosis.

Misleading testimony was claimed in the petition regarding Noah's genetic predisposition stating "to tell the jury that Noah had no genetic predisposition was simply false." [page 49] The statement must be rephrased, as every human being (and every organism with

DNA) has a genetic predisposition to whatever genetic diseases are encoded in the organism's DNA. A proper question is "Did Noah's family history contain any genetic diseases or illnesses which could contribute to the clinical presentation, clinical findings, and diagnostic considerations for explaining Noah's physical and radiographic findings?" Noah's grandfather had a history of febrile seizures. Two to five percent of children six months to 5 years of age experience a febrile seizure, and febrile seizures are the most common seizures of childhood.[8] Noah, however, did not have a febrile seizure by the definition from the American Academy of Pediatrics which defines febrile seizures as seizures that occur with fever, but in the absence of intracranial pathology, metabolic disease, or a history of non-febrile seizures.[9] Therefore, the family history of febrile seizures in Noah's grandfather had no significance in considering the genetic predisposition of Noah to a disorder causing subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusions, and injury to the corpus callosum. Similarly, a family history of Noah's father's cousins having muscular dystrophy had no significance in determining the cause for Noah's findings. Noah's findings would not be explainable even if Noah had a diagnosis of muscular dystrophy, much less a remote (father's cousins) family history of muscular dystrophy. Lastly, Noah's mother having a family history of an unspecified relative with an unspecified chromosomal abnormality who died in childhood is a significant family history, but does not therefore imply that Noah has a genetic predisposition to a specific identifiable disorder. Noah's presentation would be concerning for inherited neurodegenerative diseases of infancy (inherited disorders of brain development), however, his brain MRI showed no features even remotely suggestive of such neurodegenerative disorder. Presuming Noah remains without any such genetic disorder diagnosis, at his current age of 4 years, any sort of genetic diagnostic workup would have been unrevealing during the time of his hospitalization. The petition fails to even outline how the described family history would have altered the diagnostic evaluation or clinical care provided to Noah, or somehow would have lead the physicians caring for Noah away from the diagnostic

consideration of non-accidental injury as the cause for his clinical findings. To a reasonable degree of medical certainty, Noah's family history provides no additional information to specify the consideration of a particular genetic or non-genetic cause for his clinical findings. His clinical findings do not infer a specific genetic abnormality that would have been appropriate to investigate as an alternate diagnostic etiology for his subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusions, and injury to the corpus callosum. The testimony at trial was correct that Noah did not have any genetic predisposition to a particular disease which could explain the clinical findings at the time of his hospitalization.

Noah was treated for a purulent tracheal aspirate. He did not present to the emergency department with a fever, but was reported by the petition to have been "fussy" and "unwell" in the previous days.[page 25 and 26] The claim that "evidence of a fever and infectious growth in the lungs" should have given the jury pause in considering abusive head trauma as the cause of his findings because "there was no reason for the jury to connect his fussiness to his medical state" is not a valid argument. If Noah did have a respiratory tract infection prior to being admitted to the hospital, why was there was no clinical history of fever, cough, or respiratory distress to suggest this diagnosis by the emergency department records or the hospital records? The emergency physician documented "no fever, no fussiness, no cough" in the emergency department records. After being intubated in the emergency department at 1534, a tracheal aspirate was collected at 2342, 8 hours after intubation. Tracheitis and pneumonia are recognized complications of intubation. Noah was not diagnosed with pneumonia during his hospital stay, not because of prematurely dismissing the diagnosis, or hasty presumptions, but because he could not be definitively diagnosed with pneumonia. In pediatric critical care non-quantitative cultures are not reliable for the diagnosis of pneumonia in the severely ill patient.[10]

Traditionally the diagnosis of pneumonia is made on the clinical criteria of fever, leukocytosis, purulent sputum and infiltrate on chest radiograph. Several studies have demonstrated that the clinical criteria alone are not diagnostic. Other conditions (ARDS, SIRS, pulmonary contusion) may have similar clinical findings, yet only 50% of such patients may have pneumonia.

Noah required intubation due to coma, and persistent seizures. Acute respiratory distress syndrome can occur as a complication of the need for artificial ventilation and intubation. Noah's clinical course included acute respiratory distress syndrome as a possible cause for his abnormal chest radiographs and his respiratory symptoms following the initial extubation. Additionally, his sputum culture improved, yet the patchy abnormalities on his chest x-ray persisted. If pneumonia was continuing or worsening, then the culture should not have been improving with the worsening of abnormalities on his chest x-rays. In his discharge records the treating physicians stated that Noah was intubated in the ED, extubated the next day without difficulty except for some post-extubation stridor. This high-pitched sound (stridor) occurs from local injury or swelling to the trachea due to the presence of the plastic breathing tube. While he was treated with antibiotics for the growth of bacteria on his tracheal aspirate, this infection likely developed as a result of his intubation and ventilation.

Other causes for Noah's fever were present based upon his other clinical findings. Non-infectious fever has been described in up to half of the pediatric patients who develop fever after admission to a pediatric intensive care unit for severe traumatic brain injury.[11] Persistent generalized seizures have been described as causing fevers which last more than two days.[12] Noah was also noted to have persistent seizures requiring induction of a coma on phenobarbital, and he was electively intubated on April 25th due to the continued seizures not because of a worsening lung infection.

In summary, with no clinical history of respiratory symptoms prior to admission, with a non-quantitative tracheal aspirate culture which improved while his chest x-rays continued to have patchy abnormalities, it was more likely than not that Noah did not have a significant respiratory illness prior to his admission to the hospital. Even if Noah did have pneumonia or a respiratory infection, and even if it had developed prior to his hospitalization, the presence of the pneumonia would not account for the other clinical findings, namely it would not have explained why Noah had subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, cortical vein thrombosis, and injury to the corpus callosum. The petition states "it was important for trial counsel to focus on explaining the significance" of the cortical venous thrombosis "and the potential non-abuse causes." [page 20] This statement is predicated on an incorrect assignment to the significance of the single clinical finding of a small cortical vein thrombosis in a patient with subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, and injury to the corpus callosum, who collapses without other independent witnesses with a care-giver and presents with coma and persistent seizures.

There is no single test for Shaken Baby Syndrome. Like most medical diagnoses, determining if an infant has a syndrome or diagnosis requires a review of the available and relevant clinical history, a physical examination, and a review of the results of testing. With this information the clinician must examine the available results and assign relative merit and importance to each element. A clinician cannot weight all of the evidence equally in making a diagnosis. Certain clinical finding may have more significance in making a diagnosis than other clinical findings. For example, in diagnosis of a heart attack, the 12-lead ECG may be the most important clinical finding. Even when the ECG is normal the patient may still have the diagnosis of a heart attack based upon other clinical findings. Each clinical finding has a list of possible and probable causes. Subdural hemorrhages have multiple causes, including trauma. A physician who

is presented with an infant with an unexplained acute subdural hemorrhage may have a suspicion of non-accidental trauma and if so is thereby is mandated to report to child protective services. The diagnostic workup continues after this report to child protective services, as it did for Noah. He underwent chest x-rays, bone surveys, CT scans, an MRI, blood cultures, tracheal aspirate cultures, hematology tests including multiple tests for common and uncommon coagulation disorders, chemical tests for metabolic and electrolyte abnormalities, urine tests including culture for infection, and viral antigen testing. He had consultations from pediatric neurosurgery, pediatric neurology, pediatric forensic medicine, and pediatric physical medicine.

Despite the claims that considering "differential diagnoses was thrown out the window" [page 23, or that the physicians "failed to pursue differential diagnoses for his symptoms" [page 35], or "instead all the treating physicians simply assumed trauma and stopped looking for alternative explanations" [page 35], no list of alternative diagnoses are offered in the petition that were allegedly not considered. The only references are to "infection" [page 28] which was diagnostically evaluated as would be appropriate for an infant presenting with Noah's clinical history and findings, or to a "rebleed" [page 28] which is a conclusion without any evidence on any of Noah's tests for the present of an old hemorrhage. No reference is made to any particular test or consultation that the physicians failed to obtain in the evaluation of Noah in the hospital which could to a reasonable degree of medical certainty shown an alternate diagnostic probability to explain Noah's subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, cortical vein thrombosis, and injury to the corpus callosum.

Retinal hemorrhages have multiple causes, including trauma. Cortical vein thrombosis has multiple causes, including trauma. Noah also had injury to the corpus callosum, which has a limited list of causes, as do cortical contusions, both of which are highly

specific for trauma. Meningitis has rarely been associated with retinal hemorrhages, and even when seen the hemorrhages are often few in number and only posteriorly located. Meningitis is not associated with acute subdural hemorrhages, nor injury to the corpus callosum or cortical contusions. While meningitis could be the cause for selected features of Noah's presentation or test results, it could not explain all of his findings, and based upon Noah's hospital course to a reasonable degree of medical certainty was not present during his hospitalization. The petition suggests that oxygen deprivation may have contributed to the bleeding. [page 39] CPR was reportedly performed by Ms. Munoz at the daycare, but the pediatric physical medicine physician noted "patient has no documented history of hypoxia or arrest." The emergency department noted "babysitter gave a few rescue breaths and compressions" certainly not consistent with a length of time necessary for hypoxic brain injury. Non-medical providers such as babysitters and parents commonly interpret a seizure or unresponsiveness as an episode of cardiac arrest, even though no interruption of circulation has occurred. Noah's initial bicarbonate level was normal, and not consistent with or suggestive of a lactic acidosis from a hypoxic injury. The neurologist noted that Noah had encephalopathy which would be explained by the history of prolonged seizures when he was admitted, sedation, or trauma. The attending pediatrician noted that Noah was reported to be choking, and then he started seizing. Additionally, there was no evidence of a diffuse cortical injury as would occur from hypoxia, but instead focal abnormalities consistent with contusions and axonal injury. Thus, there was no evidence of a diffuse hypoxic brain injury, and diffuse hypoxia would not be a reasonable diagnostic consideration for Noah's findings. Finally, the cortical vein thrombosis was not the cause of Noah's symptoms nor could its presence explain the subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, and injury to the corpus callosum.

Lex parsinmoniae is principle used in medicine and is also called diagnostic parsimony. Physicians are trained when making a diagnosis look for the fewest possible causes that

will account for all of the symptoms. The petition does not offer an explanation for Noah's retinal hemorrhages, acute subdural hemorrhages, corpus callosum injury or the cortical contusions. These findings cannot be ignored. The petition is inappropriately focused on only two diagnostic features: respiratory infection and cortical vein thrombosis, and fails to consider the other diagnostic features which Noah had, apparently presuming that if the cortical vein thrombosis was the result of an infection, somehow that explains the cause for all the other findings. The other findings of subdural hemorrhages, retinal hemorrhages, bilateral occipital cortical contusions, left parietal cortical contusion, and injury to the corpus callosum are not the result of the theories posited in the petition, namely infection, dehydration, or "something" used to explain the cortical venous thrombosis. [page 21] The suggestion that "one of the strongest alternative explanations for of Noah's symptoms" [page 20] was that the cortical vein thrombosis caused a "stroke" is directly at odds with the medical evidence. A venous occlusion of a single cortical vein cannot cause the diffuse nature of Noah's findings. No widespread ischemia was found on Noah's MRI. However, repetitive rotational head trauma such as from shaking applies forces to the entirety of the head, and is therefore not a focal effect but a diffuse effect. Head trauma is a known cause of cortical vein thrombosis. Non-accidental head trauma, in particular, is a known cause of subdural hemorrhages, retinal hemorrhages, cortical contusions, and injury to the corpus callosum.

The rotational head trauma would explain the diffuse, perifalcine and vertex subdural hemorrhage with severe bilateral retinal hemorrhages and sudden coma. Despite the claims of petition, the medical literature contains hundreds of reported cases of infants with a sudden change in mental status while alone with a care-giver who are found to have severe bilateral retinal hemorrhages and acute subdural hemorrhages without any history of trauma. Based upon Noah's initial presentation and findings, non-accidental trauma was a plausible explanation for his findings but certainly not the only explanation. Further evaluation in the hospital identified additional intracranial findings such as

cortical contusions and injury to the corpus callosum which validated this initial diagnostic possibility. There is no need to implicate a complicated pathway of dehydration or infection in the lungs causing a small cortical vein thrombosis, that somehow (despite medical literature to the contrary) led to subdural hemorrhage and retinal hemorrhages without causing a large stroke, which also somehow (but never explained by the petition) caused injury to the corpus callosum and both occipital lobes which are quite distant from the location of the cortical vein thrombosis.

Repetitive rotational head trauma, such as from manual shaking is consistent with and explains this injury to the corpus callosum and the occipital lobes. Modeling of infant brain tissue has demonstrated how this injury occurs. During finite 2D modeling of repetitive oscillations of the infant brain, during anterior impact of the brain against the inner surface of the skull, the anterior lobe moves laterally with a corresponding medial movement of the posterior brain. During posterior impact a similar effect occurs. The overall effect of 8 repeated movements over two seconds creates "stretching, squashing, and end rotation generate strain concentrations around the corpus callosum and deep brain areas."[13] The end rotation of the cerebral hemispheres would occur at the occipital lobes where Noah's bilateral cortical contusions were found.

Assuming that Noah was fussy and not feeling well, as alleged in the petition, [page 19] this would be a risk factor for becoming a victim of child abuse due to frustration or anger by a care-giver as the child is difficult to console.[14,15] Reportedly others had witnessed Noah being "cranky," that he "cried a lot," and that "there was nothing I could do to stop him from crying." [page 19] Studies are showing a significant association of infant crying with child abuse. The age-specific incidence curve of hospitalized shaken baby syndrome cases has a similar starting point and shape to the age-specific incidence curve for crying.[16] A study in France found that persons who confessed to shaking an infant commonly described a desire for the infant to stop crying.[1] "In our series, all of the

perpetrators who confessed (100%) described a violent and inappropriate attack that resulted from fatigue and irritation connected with the infant's crying."

As discussed above, to a reasonable degree of medical certainty, the subdural hemorrhages, the retinal hemorrhages, cerebral contusions, and corpus callosum injury were a direct result of violent repetitive rotational head trauma such as from shaking. The seizures suffered by Noah in his presentation to the hospital were a direct result of the injury sustained to his brain. To a reasonable degree of medical certainty, the cortical vein thrombosis is a result of the trauma to the bridging vein during the trauma from the shaking. The petition offers no new diagnostic considerations which to a reasonable degree of medical certainty would explain all of Noah's clinical findings.

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Response to Petition For a Writ of Habeas Corpus
Date: January 25, 2013
Case: Trudy Munoz Rueda v Harold W Clarke
(Whitmer, Noah)

ZĒDA MEDICAL INSTITUTE

William E. Hauda II, MD

AFFIDAVIT

I, William E Hauda II MD, was an examining or treating health care provider of Noah Whitmer, whose date of birth is 29-NOV-2008. The information contained in the above is true, accurate and fully describes the nature and extent of the physical condition or injury suffered by this child.

January 25, 2013
Date

William Hauda, M.D.
Pediatric Forensic Assessment and
Consultation Team, Inova Fairfax
Hospital for Children

STATE OF Virginia :
COUNTY OF Fairfax :
TO WIT:

SUBSCRIBED and SWORN TO before me this 25th day of January 2013. In testimony whereof I have hereunto set my hand the day, month and year aforesaid.

Tesia M. Vargason
NOTARY PUBLIC

TESIA MELODY VARGASON
Notary Public
Commonwealth of Virginia
7281199
My Commission Expires Nov 30, 2013

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Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking

Catherine Adamsbaum, Sophie Grabar, Nathalie Mejean and Caroline Rey-Salmon

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Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking

OBJECTIVE: Confessions are uncommon in abusive head trauma (AHT) cases, and there is debate over whether shaking alone can cause the injuries characteristic of AHT. The objective of this article is to correlate legal statements by perpetrators with medical documentation to offer insights into the mechanism of injury.

METHODS: In this retrospective observational study we examined forensic evidence from 112 cases referred for AHT over a 7-year period. We compared 29 cases in which a perpetrator confessed to violence toward the child with 83 cases in which there was no confession. Inclusion criteria were subdural hematoma (SDH) on computed tomography and perpetrator admission of a causal relationship between the violence inflicted and the child's symptoms. Groups were compared by using Student's *t* test for age and Fisher's exact test for gender, death, fractures, retinal hemorrhages, ecchymoses, symptoms, and SDH patterns. All medical records from birth to diagnosis, imaging studies, and written investigation reports were reviewed.

RESULTS: All confessions came from forensic investigations. There was no statistically significant difference between the 2 groups for any of the variables studied. Shaking was described as extremely violent (100%) and was repeated (55%) from 2 to 30 times (mean: 10) because it stopped the infant's crying (62.5%). Impact was uncommon (24%). No correlation was found between repeated shaking and SDH densities.

CONCLUSIONS: This unique forensic case series confirms the violence of shaking. The high frequency of habitual AHT is a strong argument for reporting suspected cases to judicial authorities and helps to explain the difficulty in dating the injuries. *Pediatrics* 2010;126:546–555

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KEY WORDS
abusive head trauma, subdural hematoma, child abuse

ABBREVIATIONS
AHT—abusive head trauma
CT—computed tomography
SDH—subdural hematoma

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Abusive head trauma (AHT) is a significant cause of severe brain injury in infants, and a leading area of controversy is whether shaking alone is sufficient to cause the characteristic injuries associated with AHT.[1,2] Violent shaking is thought to subject the infant's head to acceleration-deceleration and rotational forces that create differential movement of the brain within the cranial compartment, which results in subdural, subarachnoid, and retinal hemorrhages often associated with hypoxic-ischemic injury.[3,4] Brain computed tomography (CT) is often the first examination to be used for patients with acute injury for demonstrating the subdural hematoma (SDH) that provides one of the diagnostic clues.

Because perpetrators rarely admit to inflicting an injury, however, little is actually known about exactly what happened and when.[5] Some reports have pointed to the potential for confusion in dating the brain injuries by radiologists, even those who are trained in pediatrics using both CT and MRI.[6]

Therefore, we analyzed detailed legal statements by perpetrators and correlated confessed histories with medical documentation to offer insights into the etiology of injury.

PATIENTS AND METHODS

This observational retrospective study was conducted over a 7-year period from January 2002 to May 2009. Among 112 patients diagnosed with AHT and referred to 39 different French courts for forensic investigation, we compared 29 cases (group A) in which a perpetrator confessed to violence toward the child with 83 cases (group B) in which there was no confession.

The inclusion criteria for the 112 patients were the presence of a SDH on CT scan and perpetrator conviction for AHT. The diagnosis of AHT was based on the presence of SDH with or without traumatic skin lesions, skeletal fractures, or retinal hemorrhage in the absence of accidental trauma or metabolic or infectious pathology. The initial diagnosis of AHT was made by the pediatrician who reported the case to the social and judicial authorities. The diagnosis was later confirmed by the medical experts (Drs Adamsbaum and Rey-Salmon).

The selection criteria for group A was an admission by the perpetrator of a causal relationship between the violence inflicted and the child's symptoms. Perpetrators gave detailed descriptions of events, available in writing, during the various hearings. These 29 cases were selected for the study, and perpetrator statements were correlated with the patterns of SDH found on CT.

The infants' forensic and medical records from birth to the time of diagnosis were analyzed by the authors (Drs Adamsbaum and Rey-Salmon) as part of the judicial process. As forensic medical experts, they had access to the complete medical and judicial records. All available imaging studies (CT, MRI, standard radiographs, and ultrasound) were reviewed by a senior pediatric radiologist (Dr Adamsbaum).

For each patient, SDHs were categorized on CT as occurring in a single location or in multiple separate locations and as hyperdense (homogeneous or heterogeneous including mixed density) or hypodense. In case of multiple separate locations, SDHs were categorized as (1) having the same density in all sites, either all hyperdense or all hypodense, or (2) having different densities at different locations (eg, hyperdense deep SDH [interhemispheric and/or posterior fossa]) associated with a hypodense lateral SDH (Fig 1).

The medical, biological, toxicological, and histologic data were analyzed by Drs Adamsbaum and Rey-Salmon. If necessary to draw a conclusion, they could and did request missing medical records and conduct additional etiologic analysis (hemostasis, metabolic testing) with the judge's consent.

The details of all written investigation reports were carefully studied for each patient. In particular, we analyzed the number of violent acts reported, the delay between shaking and symptoms, the behavior of the child after the violence, the mechanism of the violence inflicted, and the indication of head impact. When the perpetrator reported "multiple episodes of shaking per week," we counted it as 2 episodes per week.

A senior forensic pediatrician (Dr Rey-Salmon) performed medical examinations on all surviving children at the time of the judicial proceedings.

Groups A and B were compared by using Student's *t* test for age and Fisher's exact test for other qualitative variables such as demographics, symptoms, and lesion characteristics at presentation (gender, death, isolated vomiting, seizures, loss of consciousness, cardiopulmonary arrest, behavior changes, strabismus, presence of fractures, retinal hemorrhages, ecchymoses, and SDH patterns). A P value $<.05$ was used to denote statistical significance.

RESULTS

Description and Comparison Between the 2 Groups

Of 112 patients, 109 had 1 or more of the following, in addition to the SDH: previous or current ecchymoses; previous or current fractures; and current retinal hemorrhages (Table 1).

In 83 of 112 cases (group B), despite evidence of AHT, there was no admission of a causal relationship between the violence inflicted and the child's symptoms. In 19 of these cases, a perpetrator reported having shaken the child violently to revive him or her from

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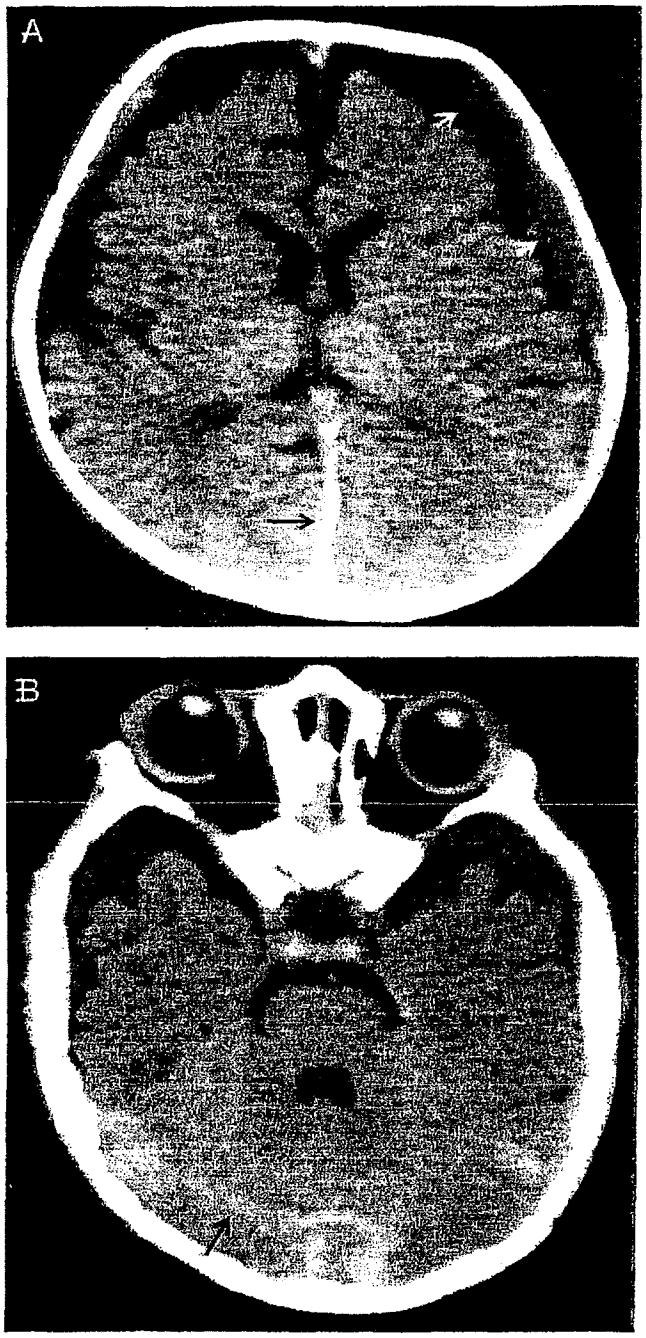

FIGURE 1
CT images of a 5-month-old infant who suffered multiple shakings over more than 1 month. Multifocal SDHs of different densities can be seen in separate locations. A, Lateral hypodensity (white arrows) associated with interhemispheric hyperdensity (black arrow); B, tentorium cerebelli hyperdensity (black arrow).

an apparent life-threatening event; in 28 cases, a minor accident was reported (short-distance fall or impact); and in 36 cases, the child's entourage did not report any particular event during the observation period.

The overall male-to-female gender ratio was 3.15 (85 boys, 27 girls). The mean age was 5.6 ± 4.8 months (range: 3 weeks to 31 months, excluding 3 outliers older than 40 months [84 and 42 months in group A and 48 months in group B]).

In the 112 patients, there were 30 (27%) fractures, 53 (47%) ecchymoses, and 99 retinal hemorrhages (88%). The most frequent symptom at presentation was seizure, which occurred in 69 of the 112 (62%) patients.

SDHs were located in multiple sites in 111 of the 112 children (99%). They were found interhemispherically (n = 107 [95.5%]), in the tentorium cerebelli (n = 96 [86%]), and in the right or left lateral spaces (n = 112 [100%]). They were hyperdense at all sites in 39 (35%), hypodense in all sites in 2 (2%), and hypodense in lateral locations and hyperdense in deep locations in 71 (63%) of these 112 children.

There was no statistically significant difference between the groups with and without detailed confessions for any of the variables studied (ie, age, gender ratio, number of deaths, main symptoms, presence of fractures, ecchymoses, retinal hemorrhages or SDH pattern [all $P > .05$] [Table 1]).

Population With Detailed Confessions (N = 29)

There were 7 girls and 22 boys (gender ratio: 1:3) aged 1 month to 7 years at the time of diagnosis (mean age: 8 months). Of these children, 27 (93%) were younger than 1 year. Nine infants (31%) had died.

In 23 of the 29 cases (79%), the major clinical symptoms that led to CT were acute, mainly seizures (n = 19 [65.5%]). Other symptoms included vomiting (n = 2), anorexia (n = 1), hip trauma (n = 1), strabismus (n = 1), and abnormal tonicity (n = 1) (Table 2).

Eleven infants (38%) had multiple ecchymoses (n = 10, including 8 cases in nonambulatory infants and 2 cases of atypical locations [such as skull, face, or trunk] in ambulatory children) or hematoma of the tongue (n = 1). Two children had a weight for age below −3 SDs at the time of diagnosis. Two

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TABLE 1 Comparison Between Group A (Full Confessions) and Group B (Without Full Confessions)

	Group A ($N = 29$)	Group B ($N = 83$)	P^a
Age, mean ± SD, mo[b]	4.7 ± 2.9	6 ± 5.5	.21
Gender ratio, male/female	22/7	63/20	1.00
Death, *n* (%)	9 (31.0)	16 (19.2)	.19
Isolated vomiting, *n* (%)	2 (6.9)	8 (9.6)	1.00
Seizures, *n* (%)	19 (65.5)	50 (60.2)	.66
Loss of consciousness, *n* (%)	2 (6.9)	7 (8.4)	1.00
Cardiopulmonary arrest, *n* (%)	2 (6.9)	12 (14.4)	.51
Behavior changes, *n* (%)[c]	2 (6.9)	2 (2.4)	.27
Strabismus, *n* (%)	1 (3.4)	0 (0.0)	.26
Macrocrania, *n* (%)	0 (0.0)	2 (2.4)	1.00
No neurologic signs, *n* (%)	1 (3.4)	2 (2.4)	1.00
Fractures, *n* (%)	11 (37.9)	19 (22.9)	.11
Skull fracture, *n* (%)	3 (10.3)	7 (8.4)	.72
Ecchymoses, *n* (%)	11 (37.9)	42 (50.6)	.23
Retinal hemorrhage, *n* (%)	24 (82.7)	75 (90.3)	.19
SDH, multiple separate locations, *n* (%)	29 (100)	82 (98.7)	1.00
Hyperdensity in all sites, *n* (%)	11 (37.9)	28 (33.7)	.82
Hypodensity in all sites, *n* (%)	2 (6.9)	0 (0.0)	.07
Hypodense lateral/hyperdense deep sites, *n* (%)	16 (55.1)	55 (66.2)	.32

Shown are symptoms and signs at presentation.

[a] Student's *t* test for age; Fisher's exact test for other variables.

[b] SD excluding 3 outliers aged >36 months (84, 42 months in group A, 48 months in group B).

[c] Including anorexia ($n = 1$, group A) and irritability ($n = 1$ group A, $n = 2$ group B).

children had been born prematurely (at 31 and 34 weeks' gestation).

Previous signs of maltreatment were found in the medical records of 8 children (27%). These signs were ecchymoses in nonambulatory children ($n = 5$) noticed from 1 week to 2 months before the acute episode, elbow fracture 5 months before the acute episode ($n = 1$), and a loss of weight under −3 SDs for age ($n = 1$). In none of these cases did these signs lead to suspicion of the diagnosis.

Ophthalmologic examination was performed on all infants, after death in 2 cases (patients 5 and 14). Funduscopy results were considered normal in 5 cases (17%) and revealed retinal hemorrhages that were bilateral in 22 cases (76%) and unilateral in 2 cases.

All but 1 of the infants had a skeletal survey; 11 (38%) had fractures at the time of diagnosis, including 3 skull fractures (Table 1). Five children had healing rib fractures that appeared to be multiple in 4 of them and single in 1 patient (patient 29); 2 children had vertebral fractures (lumbar crush fractures at L2 and L3 in patient 21 and transverse fracture of L3 together with rib fractures in patient 5). Of the 9 patients with multiple fractures, 2 presented age-different fractures (healing fracture of the clavicle and recent fracture of the femoral shaft in patient 2 and rib fracture, classic metaphyseal lesions [CMLs] of the knees and ankles, and acromion fracture in patient 17). CMLs of the knees, ankles, and elbows were seen in 3 patients (patients 17, 19, and 28) at a healing stage (periosteal appositions), and another (patient 14) presented a deformity of the distal part of the right humerus that appeared traumatic in origin. In patient 28, the distal CML of the tibia was associated with a healing spiral tibial fracture.

All infants underwent noncontrast CT scanning (inclusion criterion). The time between admission and CT ranged from 6 hours to 4 days, depending on the infant's clinical status.

In all cases, CT revealed at least 2 separate locations of SDHs. They were found in interhemispheric (27 of 29 [93%]), tentorium cerebelli (24 of 29 [83%]), and lateral right or left (29 of 29 [100%]) spaces.

The SDHs had the same density in all sites in 13 patients, either all hyperdense (homogeneous or heterogeneous) ($n = 11$; Figs 2 and 3) or all hypodense ($n = 2$). In the remaining 16 of 29 patients, the SDHs had different densities at different locations: hyperdense deep SDH (interhemispheric and/or posterior fossa) associated with a hypodense lateral SDH (Fig 1).

In addition, 22 of 29 patients presented with focal or diffuse parenchymal hypodensities on the initial CT scan.

When performed ($n = 14$), MRI confirmed the SDHs in all cases. All but 1 of the patients who underwent diffusion-weighted sequences ($n = 12$) exhibited hypoxic-ischemic injury patterns (Fig 3).

Perpetrator Statements

No statement was obtained during hospitalization. All confessions came during police custody or the judicial investigation, weeks or months after the diagnosis. The perpetrator was the father or stepfather in 13 cases (45%), the mother in 8 cases (27%), the child minder in 6 cases (21%), a teenaged brother in 1 case (patient 12), and both the mother and stepfather in the case of 1 young boy (patient 5, 7 years old) (Table 3).

All of the perpetrators described having shaken the infant violently. All the confessions included terms that denoted violence, and all the authors admitted the violence of their acts in response to the corresponding question from the court or the police inquiries. All children were taken under the arms and shaken violently, sometimes with verbal abuse. In 5 cases, a final violent impact of the infant's head on a bed was described.

The shaking was described as a single violent episode in 13 cases (45%). In 4

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TABLE 2 Characteristics of the Population ($N = 29$)

Patient No.	Gender	Age, mo	Symptom at Diagnosis	Retinal Hemorrhage	Skin Lesions	Fracture	Other	Previous Injury
1	M	2	Vomiting	0	Ecchymoses	Ribs	Loss of weight	Loss of weight
2	M	1	Hip trauma	0	0	Clavicle, femur	0	0
3	M	6	Seizures	Bilateral	0	0	0	0
4[a]	M	6	Seizures	Bilateral	Ecchymoses	Skull	0	0
5[a]	M	84	Coma	ND	Ecchymoses, burns	Ribs, lumbar transverse	Loss of weight	Ecchymoses
6	M	1	Seizures	Bilateral	Ecchymoses	0	0	0
7[a]	M	6	Seizures	Bilateral	Ecchymoses	0	0	0
8[a]	F	2	CP arrest	Bilateral	Ecchymoses	0	Premature: 31 wk gestation	0
9	F	3	Anorexia	Bilateral	0	0	0	0
10	M	8	Seizures	Left	0	0	0	0
11	M	4	Seizures	Bilateral	0	0	0	0
12	M	6	Seizures	Bilateral	0	Ribs	0	Ecchymoses
13	F	5	Seizures	Bilateral	0	0	0	Ecchymoses
14[a]	M	42	Coma	ND	Ecchymoses	Elbow, skull	0	Fracture
15	M	5	Seizures	Bilateral	Ecchymoses	0	0	Ecchymoses
16	M	5	CP arrest	Bilateral	0	0	0	Ecchymoses
17[a]	M	2	Seizures	Bilateral	Ecchymoses	Ribs, metaphyses	0	0
18	M	5	Seizures	Bilateral	0	0	0	0
19	F	4	Hypotony	0	0	Metaphyses, skull	Premature: 34 wk gestation	Ecchymoses
20	M	6	Seizures	Bilateral	0	0	0	0
21	M	2	Seizures	Bilateral	0	Vertebra	0	0
22	M	10	Strabismus	Bilateral	0	0	0	0
23	F	6	Seizures	Bilateral	0	0	0	0
24[a]	M	1	Seizures	Bilateral	Ecchymoses	0	Tongue hematoma	0
25[a]	F	1	Seizures	0	0	0	0	0
26[a]	M	11	Seizures	Bilateral	0	0	0	0
27	M	11	Seizures	Bilateral	0	0	0	0
28	M	3	Seizures	0	0	Tibia (shaft and metaphysis)	0	0
29	F	4	Vomiting	Right	0	Rib	0	0

M indicates male; F, female; CP, cardiopulmonary; ND, not determined
[a] Died.

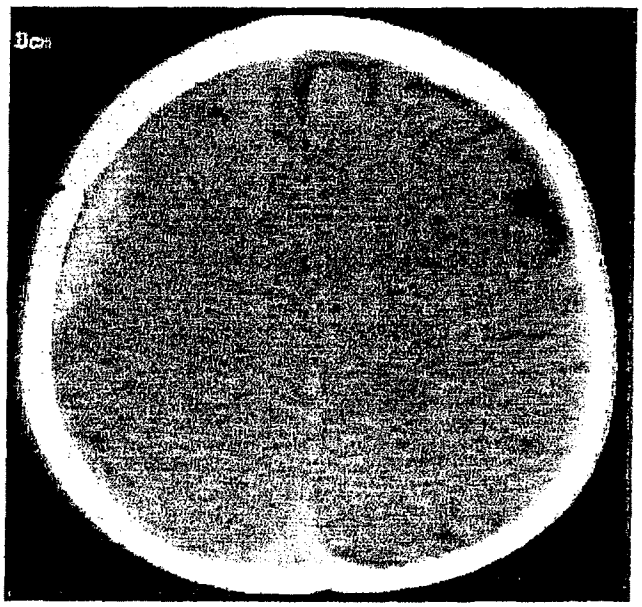

FIGURE 2
CT image of a 6-month-old infant who was shaken several times per week for more than 2 months. Right lateral and interhemispheric hyperdense SDHs can be seen, as associated with a mild mass effect on the right side.

cases, the perpetrator reported symptoms immediately after the shaking. In 6 cases, the author put the child to bed immediately after the shaking and only discovered the presence of abnormal symptoms 1.5 ($n = 4$) and 3 hours ($n = 2$) later. In 3 cases, the time delay was unclear but was <24 hours.

Repeated episodes of violent shaking were described in 16 cases (55%). The number of admitted shaking episodes ranged between 2 and 30 (mean: 10). Shaking was described as habitual (ie, daily) for several weeks or months in 6 cases. In the latter, the minimum number of episodes was estimated to be between 10 and 30. In 3 cases, the perpetrator did not give details about the number

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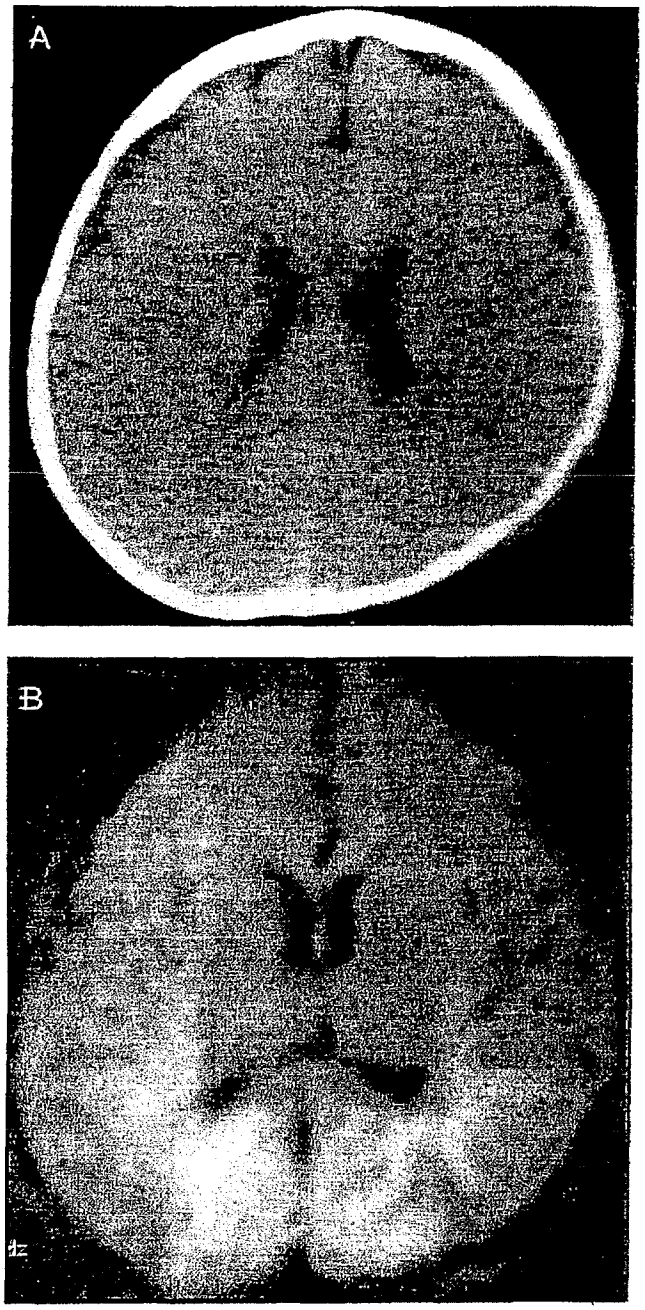

FIGURE 3
Images of a 6-month-old infant was shaken violently several times per week over 3 months. A. A CT scan shows SDH marked by subtle interhemispheric hyperdensity. Brain edema probably masks a pericerebral hypodense SDH. B. MRI (diffusion-weighted sequence) shows bilateral hyperintensities within temporo-parieto-occipital white matter related to a hypoxic-ischemic pattern. Both SDHs and hypoxic-ischemic lesions were confirmed during an autopsy and histology.

of episodes. Ten perpetrators described an immediate period of exhaustion, in which the child would "go to sleep after the shaking." All of these perpetrators reported that shaking was repeated because it stopped the infant's crying, but they did not give additional information about the final episode of shaking. The offender could not remember exactly how long the episodes of violence had been occurring.

Below are some excerpts from perpetrator statements obtained during police or judicial investigations.

"I shook him for more than 2 months, several times a week at arm's length."

"I was feeling really bad, I was at the end of my rope from not sleeping. I shook him several times a week, I don't know exactly, always at night."

"I took her by the shoulders; I shook her and I yelled."

"I was holding my daughter under the arms, and I shook her. Her head wasn't being held and was snapping back and forth."

"I thought I might have dislocated his shoulder when I shook him."

"I didn't want to choke him, but I wanted him to stop crying. I picked him up and I shook him; I threw him on the bed and he bounced on the sheet."

"When I can't calm my son I take him under the arms and, holding him firmly, I move him forward and back; I shook him several times without realizing my own strength. His head snapped back and forth from time to time. After I shake him like that, he's tired and goes to sleep. . . ."

"I shake him almost every day when I'm watching him; I can't tell you how many times; I started when he was about 4 months old."

"He was crying; it drove me crazy. I shook him . . . maybe 10 times, and threw him on the sofa."

"Once or twice I've held him at arm's length and shaken him; I've blown a fuse; over more than a month I've shaken him several times."

"I had fits of anger. She would cry; sometimes, when she did that, I'd shake her . . . I got worked up and twisted her arm; I was slapping her hard for more than 2 months."

"I hold him up in front of my face; I swing him back and forth; I'm not holding his head . . . because I'm exasperated, my movements are sometimes rough."

"I was holding him under the arms; I jostled him; I didn't shake him for long; I took him and put him down hard. There were at least 3 episodes of shaking in a little over a month; the last was harder. I had to hold him under the armpits while I was shaking him because he's too heavy (5 kg)."

"I shook her so she'd be quiet, it lasted maybe 5 minutes; I was exasperated; I shook her up and down, in front of me, without holding her against me; I was shaking her hard; I was crying just like she was, and I was worked up."

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TABLE 1 Imaging and Statements ($N = 29$)

Patient No.	Perpetrator	Delay of Symptoms	Impact	Shakings	Multifocal SDH Density	MRI, Day No.	Parenchyma	Autopsy/Histology
1	Father	—	—	Multiple	Hyperdense and hypodense	—	Normal	0
2	Father	—	—	Multiple	Hyperdense and hypodense	—	Porencephaly	0
3	Child minder	—	—	>15 over 2 mo	Hyperdense	—	Hypoxic ischemic	0
4[a]	Mother	—	—	>30 over 3 mo	Hyperdense	2	Hypoxic ischemic	SDH
5[a]	Mother and stepfather	—	yes	Single	Hyperdense	—	Hypoxic ischemic	SDH, hypoxic ischemic
6	Father	Immediate	—	Single	Hyperdense and hypodense	—	Hypoxic ischemic	0
7[a]	Mother	—	—	Single	Hyperdense	—	Hypoxic ischemic	SDH, contusions, hypoxic ischemic
8[a]	Father	—	—	At least 2 over 1 month	Hyperdense and hypodense	—	Hypoxic ischemic	SDH, cervical cord hematoma
9	Mother	—	—	At least 4 over 2 mo	Hyperdense and hypodense	—	Normal	0
10	Child minder	<1.5 h	—	Single	Hyperdense	5	Hypoxic ischemic	0
11	Father	Immediate	—	Single	Hyperdense		Hypoxic ischemic	0
12	Teenaged brother	—	yes	3 times	Hyperdense and hypodense	13	Hypoxic ischemic	0
13	Father	—	—	Multiple	Hyperdense and hypodense	—	Hypoxic ischemic	0
14[a]	Mother	Immediate	yes	Single	Hyperdense	—	Hypoxic ischemic	SDH, contusions
15	Father	—	—	>30 over 1 mo	Hypodense	—	Normal	0
16	Father	Immediate	—	Single	Hyperdense and hypodense	1	Hypoxic ischemic	0
17[a]	Father	—	yes	At least 10	Hyperdense and hypodense	3	Hypoxic ischemic	SDH, hypoxic ischemic
18	Father	—	—	At least 2 over 1 mo	Hyperdense and hypodense	—	Normal	0
19	Mother	—	—	>15 over 2 mo	Hypodense	2	Hypoxic ischemic	0
20	Child minder	—	—	At least 3	Hyperdense and hypodense	1	Hypoxic ischemic	0
21	Mother	<3 h	—	Single	Hyperdense and hypodense	0	Hypoxic ischemic	0
22	Stepfather	—	—	>10	Hyperdense and hypodense	2	Normal	0
23	Child minder	<1.5 h	—	Single	Hyperdense and hypodense	2	Hypoxic ischemic	0
24[a]	Father	—	—	Single	Hyperdense	—	Hypoxic ischemic	SDH, contusions, edema
25[a]	Mother	<1 h	—	Single	Hyperdense and hypodense	—	Hypoxic ischemic	0
26[a]	Child minder	<1 h	yes	Single	Hyperdense	5	Hypoxic ischemic	SDH
27	Child minder	<3 h	—	Single	Hyperdense	0	Hypoxic ischemic	0
28	Father	—	—	3 over 1 mo	Hyperdense	2	Hypoxic ischemic	0
29	Mother	—	—	3 over 3 wk	Hyperdense and hypodense	1	Normal	0

— indicates that data were not available.
[a] Died.

Correlations between Perpetrator Statements and Head Imaging

Five perpetrators admitted head impact, and the child died in 4 of these cases. One of these children (patient 14) was found to have a skull fracture. Two other patients (patients 4 and 19) had a skull fracture, although there was no admission of head impact, which suggests incomplete confessions (Table 3).

No correlation was found between repetitive shaking and SDH densities.

Sixteen patients were reported to have had recurrent multiple shakings. SDHs had different densities at different locations in 11 of these infants (Fig 1) and had the same density in 5. Of the latter, 3 had hyperdense homogeneous or heterogeneous SDHs (Figs 2 and 3) and 2 had hypodense SDHs.

On the other hand, 16 patients had SDHs of different densities at different locations. In these cases, 11 of the perpetrators admitted multiple shaking episodes, and 5 reported only a single violent shaking episode.

Thirteen patients exhibited SDHs of the same density in all sites: all hyperdense ($n = 11$) or all hypodense ($n = 2$). Five of the perpetrators clearly described repeated shakings. Among these cases, the SDHs were all hypodense in 2 cases.

Pathology data were available in 8 of 9 fatal cases and confirmed the presence of SDHs in all of the patients. Contusions and/or hypoxic-ischemic injuries were described for 6 patients without details about dating. Of importance is the presence of a cervical hematoma in patient 8, who did not undergo MRI.

DISCUSSION

To the best of our knowledge, this is the only case series with descriptions of confessions of forensic origin in the

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medical literature. Confessions are uncommon. Not only do perpetrators fail to acknowledge the event, but the duration of the judicial proceedings after reporting renders access to statements impossible outside of an expert medical opinion. Our results confirm the difficulties, because this series of 29 cases was the fruit of 7 years' analysis of 112 medicolegal cases of AHT.

The group of 29 infants studied displayed the classic features of AHT: multiple sites of SDHs and hypoxic-ischemic lesions, male predominance, young age, and acute signs or history of poor feeding, vomiting, or skeletal injuries.[3,7–11] There was no significant difference in mean age, gender ratio, frequency of mortality, main symptoms at presentation (vomiting, loss of consciousness, cardiopulmonary arrest, etc), ecchymoses, fractures, or retinal hemorrhages between the group with full confessions ($n = 29$) and the group without full confessions ($n = 83$) (Table 1). Because of the retrospective and unique character of the study, no power calculations were performed before beginning the study. The number of children included in the study, therefore, may have been too small to detect differences between the 2 groups. However, if these 2 groups were similar, the causal mechanism may well have been the same despite the incomplete or absent confessions. For the purposes of this study, "confession" was defined as the admission by a perpetrator of a causal relationship between the violence inflicted and the child's symptoms. In the group without confessions, admissions were of violent shaking in an attempt to revive the child from an apparent life-threatening event or minor accident (57%) or even no particular event (43%). Because detailed confessions are uncommon, it is important to focus on the information provided.

Analysis of this series of confessions highlights several basic points. First, it confirms the violence of the causal acts and, thus, the relevance of the American Academy of Pediatrics' definition: "The act of shaking leading to [AHT] is so violent that individuals observing it would recognize it as dangerous and likely to kill the child."[3] In our series, all of the perpetrators who confessed (100%) described a violent and inappropriate attack that resulted from fatigue and irritation connected with the infant's crying.

One of the most important points in this article is the role of shaking in the etiology of these injuries. As the excerpts show, all of the perpetrator statements obtained during judicial or police investigations (containing written, detailed descriptions of events) described shaking. This unique series of confessions confirms the pathogenic nature of shaking in and of itself, even without final impact.[12] We have provided excerpts from perpetrator statements to avoid interpretation.[13]

On the basis of the presence of a skull fracture or perpetrator statement, there was head impact in only a few of the cases (7 of 29). Of interest is that 2 children had a skull fracture without the perpetrator describing head impact, which suggests that the confessions were incomplete.

The main limitation of the study is that perpetrator admissions are not scientific evidence; however, they provide information that is invaluable to our understanding. Even in this context of written legal statements, some admissions are likely to be incomplete or minimized. Likewise, a single admitted episode of shaking may only be a part of the story.

The admissions of the perpetrators highlight the frequency of repeated violent shaking (55%). Shaking may be repeated on a daily basis over several weeks or months, as 6 of the perpetrators clearly reported. The estimated number of episodes ranged from 10 to 30 episodes of shaking, which is probably an underestimation, because we have assumed only 2 episodes per week when the perpetrator reported "several times" of shaking per week. Knowing that shakings are often multiple and repeated over time helps explain why it is inaccurate to date the lesions with brain imaging, CT, or even MRI.[6]

Because there was no association between SDH densities and the number of episodes of abuse, it seems clear that CT should not be used to determine chronicity of abuse. An all-hyperdense multifocal SDH was seen in 31% of admitted repetitive shakings. A hyperdense SDH can be homogeneous or heterogeneous (mixed density pattern) and may vary from one day to another.[6] Although our results did not reveal any statistically significant association between the SDH patterns on CT and the admissions of single or multiple shakings, this could be a result of the study's lack of power. A subtle hypodense SDH pattern may also be the result of AHT with habitual repetitive, violent shaking, as 2 of the statements clearly indicated. It is important to stress that a hypodense SDH may be misinterpreted as benign "external hydrocephalus." Thus, this pattern should prompt clinicians to look for bruises and/or previous unexplained symptoms.[9] In particular, a number of injuries might have been prevented if the significance of bruising in these young infants had been recognized.

Why is shaking so often repetitive? The perpetrators' statements offer an explanation. Shaking is effective because it stops the infant's crying, and he or she "goes to sleep after being shaken" (62.5%). This exhaus-

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tion reported after shaking may well be considered an immediate symptom, similar to those cited in previous reports.[12,14] In our study, in 4 cases of single shaking, the perpetrator clearly indicated immediate symptoms after the shaking. In the other cases, the timing was unclear because the child was put to bed immediately after the shaking, with symptoms discovered after a delay that was usually <3 hours. This period of "exhaustion" that occurred immediately after shaking may be a symptom of hypoxic-ischemic injuries in some of the patients. To date, hypoxic-ischemic injury is not completely understood and is probably a result of complex factors (concussion, edema, axonal and/or brainstem injury, concomitant strangulation, etc) or other causes of global neurologic dysfunction that may result from shaking.[15–17]

No admission was made during the infants' hospitalization. All the declarations came from legal statements (police custody and investigations), sometimes after a subsequent forensic investigation. Although Starling et al[14] reported cases in which the shaking was admitted in the hospital, it seems crucial that the medical corps quickly report any suspicion of AHT to the social and judicial authorities to break the vicious cycle of shaking. The main goal is to take appropriate action with regard to the offender and prevent new episodes of violence against the child, siblings, or even other children in the neighborhood.[18,19] With early identification of shaken infants, families can be offered adequate social interventions.

Physicians' decision to report may conflict with their duty to maintain the confidentiality of the doctor-family relationship.[20] However, we have not only an ethical but also a legal responsibility to report suspected abuse that supersedes any confidentiality obligation.

In France, failure to act in cases in which one suspects child abuse is considered a violation of the "duty to rescue."[21] However, physicians are not responsible for determining who injured the child; that is the job of the police.

This case series confirms that the majority of the perpetrators are fathers and stepfathers (14 of 29).[22,23] This should be interpreted with caution, however, because we do not know whether such perpetrators are violent more often or simply confess more often.

Collaboration between radiologists and clinicians is crucial for both diagnosis and prognosis in cases of child abuse.[24] Expert conclusions often have a decisive role in court decisions, and a recent study focused on the wide variability between experts.[25] Knowledge of the highly repetitive nature of AHT, the difficulty in dating lesions, and confirmation of the violent shaking involved should all help with objective interpretation.

Another limitation of this retrospective study is that a number of patients did not undergo MRI in the acute period. In France, brain CT is often the initial examination used for patients with acute injury or illness, and MRI is not yet routinely performed if the diagnosis of AHT is not in doubt and if there is no question of surgery. There are several reasons for this, including limited availability of MRI in some hospitals and unstable condition of the child in the ICU.

CONCLUSIONS

AHT is frequently related to violent shaking, the repetitive nature of which—explained by the immediate effect of shaking on crying—has been underestimated. The high frequency of habitual AHT is a strong argument for reporting suspected cases to judicial authorities and helps explain the difficulty in dating the injuries. Because CT has limitations, we recommend using MRI in addition to detect acute hypoxic-ischemic injuries as soon as the diagnosis of AHT is suspected.[4,5,26] These data should help better protect infants suspected of having AHT, and thorough police investigation will best determine the chronicity of abuse.

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Hail the Rise—Hope For The Fall—of Caesarean Births!: The New York Times *(Grady D, March 23, 2010) recently reported that the United States has reached its highest Caesarean section rate ever—32%, making it the most common operation in American hospitals as of 2007, the most recent year for which data are available. Experts remain concerned that the rate has been climbing steadily year after year since 1996 and believe the operation is being performed too often putting women at unnecessary risk. According to Dr George A. Macones, chair of obstetrics and gynecology at Washington University in St Louis, "What we're worried about is, the Caesarean section rate is going up, but we're not improving the health of babies being delivered or of moms." In addition, hospital charges for Caesareans are more than double that for vaginal deliveries. The increase affects women of all ages, races, and ethnic groups in all 50 states with the highest percentages in New Jersey and Florida and the lowest in Utah and Alaska. Whether this rate will fall remains to be seen.*

Noted by JFL, MD

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Abusive Head Trauma: Judicial Admissions Highlight Violent and Repetitive Shaking

Catherine Adamsbaum, Sophie Grabar, Nathalie Mejean and Caroline Rey-Salmon

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to create "win-win" relationships. By extension, critics of competition maintain that the NHS should do the same. These developments have been reinforced by concerns about the increase in management costs associated with the introduction of competition.

Estimates suggest that the NHS reforms may have resulted in up to £1bn extra being spent on administration, although changes in definitions make it difficult to be precise. This is because of the need to employ staff to negotiate and monitor contracts and to deal with the large volumes of paperwork involved in the contracting system. Ministers have responded to these concerns by streamlining the organisation of the NHS and introducing tight controls over management costs. They have also encouraged the use of long term contracts in order to reduce the transaction costs of the new arrangements.

Out of the ashes of competition has arisen a different policy agenda. This owes less to a belief in market forces than a desire to use the NHS reforms to achieve other objectives. The current agenda centres on policies to improve the health of the population, give greater priority to primary care, raise standards through the patient's charter, and ensure that medical decisions are evidence based. These policies hinge on effective planning and coordination in the NHS and all have been made more salient by the separation of purchaser and provider roles on which the reforms are based.

In particular, the existence of health authorities able to take an independent view of the population's health needs without being beholden to particular providers has changed the way in which decisions are made. To this extent the organisational changes introduced in 1991 have served to refocus attention on those whom the NHS exists to serve, even though the effects were neither anticipated nor intended when the reforms were designed. Like a potter moulding clay, only in the process of creation has the shape of the product become apparent. The effect of this policy shift has been to open up common ground between Labour and the Conservatives, notwithstanding the differences that remain.

Yet before the obituary of competition is written, the consequences of a return to planning need to be thought through. The NHS was reformed precisely because the old command and control system had failed to deliver acceptable improvements in efficiency and quality, and the limitations of planning must also be acknowledged. While competition as a reforming strategy may have had its day, there are nevertheless elements of this strategy which are worth preserving. Not least, the stimulus to improve performance which arises from the threat that contracts may be moved to an alternative provider should not be lost. The middle way between planning and competition is a path called contestability. This recognises that health care requires cooperation between purchasers and providers and the capacity to plan developments on a long term basis. At the same time, it is based on the premise that performance may stagnate unless there are sufficient incentives to bring about continuous improvements. Some of these incentives may be achieved through management action or professional pressure, and some may derive from political imperatives.

In addition, there is the stimulus to improve performance which exists when providers know that purchasers have alternative options. This continues to be part of the psychology of NHS decision making, even though ministers seem reluctant to use the language of markets. It is, however, a quite different approach than competitive tendering for clinical services, which would expose providers to the rigours of the market on a regular basis.

The essence of contestability is that planning and competition should be used together, with contracts moving only when other means of improving performance have failed. Put another way, in a contestable health service it is the possibility that contracts may move that creates an incentive within the system, rather than the actual movement of contracts. Of course for this to be a real incentive then contracts must shift from time to time, but this is only one element in the process and not necessarily the most important. As politicians prepare their plans for the future it is this path that needs to be explored.

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Evidence based medicine: what it is and what it isn't

It's about integrating individual clinical expertise and the best external evidence

Evidence based medicine, whose philosophical origins extend back to mid-19th century Paris and earlier, remains a hot topic for clinicians, public health practitioners, purchasers, planners, and the public. There are now frequent workshops in how to practice and teach it (one sponsored by the *BMJ* will be held in London on 24 April); undergraduate[1] and postgraduate[2] training programmes are incorporating it[3] (or pondering how to do so); British centres for evidence based practice have been established or planned in adult medicine, child health, surgery, pathology, pharmacotherapy, nursing, general practice, and dentistry; the Cochrane Collaboration and Britain's Centre for Review and Dissemination in York are providing systematic reviews of the effects of health care; new evidence based practice journals are being launched; and it has become a common topic in the lay media. But enthusiasm has been mixed with some negative reaction.[4-6] Criticism has ranged from evidence based medicine being old hat to it being a dangerous innovation, perpetrated by the arrogant to serve cost cutters and suppress clinical freedom. As evidence based medicine continues to evolve and adapt, now is a useful time to refine the discussion of what it is and what it is not.

Evidence based medicine is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of evidence based medicine means integrating individual clinical expertise with the best available external clinical evidence from systematic research. By individual clinical expertise we mean the proficiency and judgment that individual clinicians acquire through clinical experience and clinical practice. Increased expertise is reflected in many ways, but especially in more effective and efficient diagnosis and in the more thoughtful identification and compassionate use of individual patients' predicaments, rights, and preferences in making clinical decisions about their care. By best available external clinical evidence we mean clinically relevant research, often from the

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basic sciences of medicine, but especially from patient centred clinical research into the accuracy and precision of diagnostic tests (including the clinical examination), the power of prognostic markers, and the efficacy and safety of therapeutic, rehabilitative, and preventive regimens. External clinical evidence both invalidates previously accepted diagnostic tests and treatments and replaces them with new ones that are more powerful, more accurate, more efficacious, and safer.

Good doctors use both individual clinical expertise and the best available external evidence, and neither alone is enough. Without clinical expertise, practice risks becoming tyrannised by evidence, for even excellent external evidence may be inapplicable to or inappropriate for an individual patient. Without current best evidence, practice risks becoming rapidly out of date, to the detriment of patients.

This description of what evidence based medicine is helps clarify what evidence based medicine is not. Evidence based medicine is neither old hat nor impossible to practice. The argument that "everyone already is doing it" falls before evidence of striking variations in both the integration of patient values into our clinical behaviour[7] and in the rates with which clinicians provide interventions to their patients.[8] The difficulties that clinicians face in keeping abreast of all the medical advances reported in primary journals are obvious from a comparison of the time required for reading (for general medicine, enough to examine 19 articles per day, 365 days per year[9]) with the time available (well under an hour a week by British medical consultants, even on self reports[10]).

The argument that evidence based medicine can be conducted only from ivory towers and armchairs is refuted by audits from the front lines of clinical care where at least some inpatient clinical teams in general medicine,[11] psychiatry (J R Geddes *et al,* Royal College of Psychiatrists winter meeting, January 1996), and surgery (P McCulloch, personal communication) have provided evidence based care to the vast majority of their patients. Such studies show that busy clinicians who devote their scarce reading time to selective, efficient, patient driven searching, appraisal, and incorporation of the best available evidence can practice evidence based medicine.

Evidence based medicine is not "cookbook" medicine. Because it requires a bottom up approach that integrates the best external evidence with individual clinical expertise and patients' choice, it cannot result in slavish, cookbook approaches to individual patient care. External clinical evidence can inform, but can never replace, individual clinical expertise, and it is this expertise that decides whether the external evidence applies to the individual patient at all and, if so, how it should be integrated into a clinical decision. Similarly, any external guideline must be integrated with individual clinical expertise in deciding whether and how it matches the patient's clinical state, predicament, and preferences, and thus whether it should be applied. Clinicians who fear top down cookbooks will find the advocates of evidence based medicine joining them at the barricades.

Some fear that evidence based medicine will be hijacked by purchasers and managers to cut the costs of health care. This would not only be a misuse of evidence based medicine but suggests a fundamental misunderstanding of its financial consequences. Doctors practising evidence based medicine will identify and apply the most efficacious interventions to maximise the quality and quantity of life for individual patients; this may raise rather than lower the cost of their care.

Evidence based medicine is not restricted to randomised trials and meta-analyses. It involves tracking down the best external evidence with which to answer our clinical questions. To find out about the accuracy of a diagnostic test, we need to find proper cross sectional studies of patients clinically suspected of harbouring the relevant disorder, not a randomised trial. For a question about prognosis, we need proper follow up studies of patients assembled at a uniform, early point in the clinical course of their disease. And sometimes the evidence we need will come from the basic sciences such as genetics or immunology. It is when asking questions about therapy that we should try to avoid the non-experimental approaches, since these routinely lead to false positive conclusions about efficacy. Because the randomised trial, and especially the systematic review of several randomised trials, is so much more likely to inform us and so much less likely to mislead us, it has become the "gold standard" for judging whether a treatment does more good than harm. However, some questions about therapy do not require randomised trials (successful interventions for otherwise fatal conditions) or cannot wait for the trials to be conducted. And if no randomised trial has been carried out for our patient's predicament, we must follow the trail to the next best external evidence and work from there.

Despite its ancient origins, evidence based medicine remains a relatively young discipline whose positive impacts are just beginning to be validated,[12,13] and it will continue to evolve. This evolution will be enhanced as several undergraduate, postgraduate, and continuing medical education programmes adopt and adapt it to their learners' needs. These programmes, and their evaluation, will provide further information and understanding about what evidence based medicine is and is not.

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For details of the international conference on evidence based medicine to be held in London on Wednesday 24 April 1996, contact the BMA/BMJ Conference Unit, telephone 0171 383 6605, fax 0171 383 6663.

FED APP 296

IN THE CIRCUIT COURT OF FAIRFAX COUNTY
CALENDAR CONTROL FORM

PLEASE PRINT AND USE BLACK INK ONLY

Commonwealth versus Truoy Eliana Munoz Rueda

CASE NUMBER FE 2009 - 1289

Criminal	Juvenile	Law	Chancery	Fiduciary
X				

PARTY REQUESTING CONTINUANCE (Please check)
Counsel for Plaintiff _____ Counsel for Defendant X
Counsel for Commonwealth X Pro Se Plaintiff/Defendant _____

REASON: This is a felony Child Abuse Case. Grand Jury was July 20, 2009. Trial was set by J. Tobr court for August 12, 2009. This case is medical records intensive with both sides having expert witnesses. It is taking considerable time getting records from John Hopkins, MD; Childrens Hosp, D.C.; FAIRFAX INOVA

IF THERE HAS BEEN A PREVIOUS MISTRIAL, PLEASE INDICATE WHICH JUDGE HEARD THE PREVIOUS TRIAL. _____

NAME OF ATTORNEY(S) FOR PLAINTIFF/COMMONWEALTH

Gregory O. Holt
Please print name

Signature

Telephone #: (703) 246.2776 Is your client currently incarcerated: Yes _____ No _____

NAME OF ATTORNEY(S) FOR DEFENDANT

Guillermo Uriarte
Please print name

Signature

Telephone #: (703) 998-0000 948-0000 Is your client currently incarcerated: Yes ✓ No _____

TO BE COMPLETED BY CALENDAR CONTROL JUDGE

GRANTED _____ DENIED _____

August 12, 2009	10/26, 27+28		2-3 days
Old Trial Date	New Trial Date	Judge Assigned	Time Estimate

SET FOR: TRIAL _____ JURY X 2-3 days NO JURY _____

INTERPRETER ASSIGNED: YES _____ NO ✓ NONE NEEDED _____

MOTIONS _____ * Need Spanish

*If set for a FRIDAY, indicate which Motion Docket.

_____ 9:00 a.m. WJ
_____ 10:00 a.m. 2-Week Motion
_____ 11:30 a.m. 2-Week Motion
_____ 9:00 a.m. W/OJ
_____ 10:00 a.m. Regular
_____ 11:30 a.m. Regular

CALENDAR CONTROL JUDGE

7/24/09
DATE

EXHIBIT C

CH

Cindy CW Nancy

IN THE CIRCUIT COURT OF FAIRFAX COUNTY
CALENDAR CONTROL FORM

PLEASE PRINT AND USE BLACK INK ONLY

Commonwealth of Virginia versus Trudy Munoz Rueda

CASE NUMBER

Criminal	Juvenile	Law	Chancery	Fiduciary
✓			FE2009-1289	

PARTY REQUESTING CONTINUANCE (Please check)

Counsel for Plaintiff _____ Counsel for Defendant ✓

Counsel for Commonwealth _____ Pro Se Plaintiff/Defendant _____

REASON: Expert Witness for defense is not available for Trial, on October 26-28.

IF THERE HAS BEEN A PREVIOUS MISTRIAL, PLEASE INDICATE WHICH JUDGE HEARD THE PREVIOUS TRIAL. _____

NAME OF ATTORNEY(S) FOR PLAINTIFF/COMMONWEALTH

Gren Holt — Signature

Please print name

Telephone #: (___) _____ Is your client currently incarcerated: Yes _____ No _____

NAME OF ATTORNEY(S) FOR DEFENDANT

Guillermo Uriarte — Signature

Please print name

Telephone #: (___) _____ Is your client currently incarcerated: Yes X No _____

TO BE COMPLETED BY CALENDAR CONTROL JUDGE

GRANTED X DENIED _____

Old Trial Date	New Trial Date	Judge Assigned	Time Estimate
10.26.09	12/7/09		3 days

SET FOR: TRIAL X JURY X NO JURY _____

INTERPRETER ASSIGNED: YES X NO _____ NONE NEEDED Spanish

Δ counsel waives speedy trial

MOTIONS _____*

*If set for a FRIDAY, indicate which Motion Docket.

_____ 9:00 a.m. WJ
_____ 10:00 a.m. 2-Week Motion
_____ 11:30 a.m. 2-Week Motion
_____ 9:00 a.m. W/OJ
_____ 10:00 a.m. Regular
_____ 11:30 a.m. Regular

BAW

CALENDAR CONTROL JUDGE

7/9/09

DATE

FED APP 298

Cindy cc Nancy

IN THE CIRCUIT COURT OF FAIRFAX COUNTY
CALENDAR CONTROL FORM

PLEASE PRINT AND USE BLACK INK ONLY

Commonwealth versus Trudy Elana Munoz Rueda

CASE NUMBER FE 2009-1289

Criminal	Juvenile	Law	Chancery	Fiduciary
X				

PARTY REQUESTING CONTINUANCE (Please check)
Counsel for Plaintiff _____ Counsel for Defendant _____
Counsel for Commonwealth X Pro Se Plaintiff/Defendant _____

REASON: AN ESSENTIAL WITNESS FOR THE COMMONWEALTH WILL BE OUT OF TOWN (IN SOUTH AFRICA) ON CURRENT TRIAL DATE. SITE SCHEDULED TRIP PRIOR TO RECEIVING WITNESS SUBPOENA. DEFENDANT HAS HAD 2 PRIOR CONTINUANCES FOR ITS EXPERT WITNESS.

IF THERE HAS BEEN A PREVIOUS MISTRIAL, PLEASE INDICATE WHICH JUDGE HEARD THE PREVIOUS TRIAL. _____

NAME OF ATTORNEY(S) FOR PLAINTIFF/COMMONWEALTH

Gregory O'Holt — Signature
Please print name
Telephone #: (703) 246-2776 Is your client currently incarcerated: Yes _____ No _____

NAME OF ATTORNEY(S) FOR DEFENDANT

Guillermo Uriarte — Signature
Please print name
Telephone #: (703) 998-0000 Is your client currently incarcerated: Yes X No _____

TO BE COMPLETED BY CALENDAR CONTROL JUDGE

GRANTED ✓ DENIED _____

Old Trial Date	New Trial Date	Judge Assigned	Time Estimate
Dec 7, 2009	1-11-10		3 day

SET FOR: TRIAL _____ JURY X 3 DAYS NO JURY _____

INTERPRETER ASSIGNED: YES X Spanish NO _____ NONE NEEDED _____

MOTIONS _____ *

*If set for a FRIDAY, indicate which Motion Docket.

_____ 9:00 a.m. WJ
_____ 10:00 a.m. 2-Week Motion
_____ 11:30 a.m. 2-Week Motion
_____ 9:00 a.m. W/OJ
_____ 10:00 a.m. Regular
_____ 11:30 a.m. Regular

CALENDAR CONTROL JUDGE — DATE 10-22-09

FED APP 299

VIRGINIA

IN THE CIRCUIT COURT FOR THE COUNTY OF FAIRFAX

TRUDY MUÑOZ RUEDA,	)	
Petitioner,	)	
	)	
v.	)	**Case No. 2012-17074**
	)	
	)	
HAROLD W. CLARKE, Director,	)	
Virginia Department of Corrections,	)	
Respondent.	)	

EXHIBITS TO
REPLY IN OPPOSITION TO MOTION TO DISMISS

Ex. 1 - Barnes, et al., *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, 18 , 18 TOP MAG RESON IMAGING 53.

Ex. 2 – Second Affidavit of Dr. Patrick Barnes.

Ex. 3 – Three photographs of Noah Whitmer's head.

Ex. 4 – Second Kearney Affidavit

Ex. 1

Barnes, et al., *Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics*, 18 , 18 TOP MAG RESON IMAGING 53.

Imaging of the Central Nervous System in Suspected or Alleged Nonaccidental Injury, Including the Mimics

Patrick D. Barnes, MD and Michael Krasnokutsky, MD

Abstract: Because of the widely acknowledged controversy in nonaccidental injury, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine. Children with suspected nonaccidental injury versus accidental injury must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate pattern of injury and timing issues and to consider the mimics of abuse. All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only central nervous system injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.

Key Words: child abuse, computed tomography, magnetic resonance imaging, nonaccidental injury, nonaccidental trauma

(*Top Magn Reson Imaging* 2007;18:53–74)

Traumatic central nervous system (CNS) injury is reportedly the leading cause of childhood morbidity and mortality in the United States, resulting in about 100,000 emergencies annually and half the deaths from infancy through puberty.[1–5] The major causes are accidental injuries (AIs) and include falls, vehicular accidents, and recreational mishaps. However, nonaccidental, inflicted, or intentional trauma is said to be a frequent cause, with peak incidence at the age of about 6 months and accounting for about 80% of the deaths from traumatic brain injury in children younger than 2 years. Nonaccidental injury (NAI)—or nonaccidental trauma (NAT)—is the more recent terminology applied to the traditional labels *child abuse*, *battered child syndrome*, and *shaken baby syndrome* (SBS).[4,5] A modern restatement of the definition of SBS is that it represents a form of physical NAI to infants characterized by "the triad" of (1) subdural hemorrhage (SDH), (2) retinal hemorrhage (RH), and (3) encephalopathy (ie, diffuse axonal injury [DAI]) occurring in the context of inappropriate or inconsistent history and commonly accompanied by other apparently inflicted injuries.[6] The short-term life-threatening presentations and long-term outcomes have become a major concern in health care, dating back to the original reports of Kempe,[7] Caffey,[8] and Silverman.[9] Later reports on the incidence rate of CNS trauma in alleged NAI estimate a range of 7% to 19%.[4,5]

However, a number of reports from multiple disciplines have challenged the evidence base (ie, quality of evidence [QOE] analysis) for NAI/SBS as the cause in all cases of the triad.[4,5,10] Such reports indicate that the triad may also be observed in AI (including those associated with short falls, lucid interval, and rehemorrhage) and in nontraumatic or medical conditions. These are the "mimics" of NAI that often present as acute life-threatening events (ALTE). This includes hypoxia-ischemia (eg, apnea, choking, respiratory or cardiac arrest), ischemic injury (arterial vs venous occlusive disease), seizures, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, and metabolic or connective tissue disorders. Many cases seem multifactorial and involve a combination or sequence of contributing events or conditions.[4,5,10] For example, an infant is dropped and experiences a head impact with delayed seizure, choking spell, or apnea, and then undergoes a series of prolonged or difficult resuscitations, including problematic airway intubation with subsequent hypoxic-ischemic brain injury. Another example is a young child with ongoing infectious illness, fluid-electrolyte imbalance, and coagulopathy, and then experiences seizures, respiratory arrest, and resuscitation with hypoxic-ischemic injury.

Often, the imaging findings are neither characteristic of nor specific for NAI. Because of the widely acknowledged controversy in NAI, the radiologist involved in such cases must be thoroughly familiar with the imaging, clinical, surgical, pathological, biomechanical, and forensic literature from all perspectives and with the principles of evidence-based medicine (EBM).[4,5,10] Children with suspected NAI versus AI must not only receive protective evaluation but also require a timely and complete clinical and imaging workup to evaluate the pattern of injury and timing issues and to consider the mimics of abuse.[4,5,10] All imaging findings must be correlated with clinical findings (including current and past medical record) and with laboratory and pathological findings (eg, surgical, autopsy). The medical and imaging evidence, particularly when there is only CNS injury, cannot reliably diagnose *intentional* injury. Only the child protection investigation may provide the basis for *inflicted* injury in the context of *supportive* medical, imaging, biomechanical, or pathological findings.[4,5,10]

From the Stanford University Medical Center, Stanford, CA.
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MECHANISMS AND MANIFESTATIONS OF TRAUMATIC CNS INJURY

The spectrum of CNS injury associated with trauma (AI or NAI) has been classified into primary versus secondary, focal versus diffuse, and acute versus chronic categories.[4,5,10,11] The primary injury is immediate, irreversible, and is the direct result of the initial traumatic force (eg, contusion, shear injury). Secondary injury denotes the reactive phenomena that arise from or are associated with the primary injury (eg, swelling, hypoxia-ischemia, herniation). Direct contact or impact phenomena produce localized cranial distortion or deformation and thus produce *focal* injury (eg, fracture [Fx], contusion, epidural hematoma [EDH]). Accidental injury is said to be typically associated with this mechanism and result (Fig. 1). Although reported also in cases of NAI, it has been stated that impact injury, with the exception of EDH, is usually not life threatening.

It is *indirect* trauma (ie, independent of skull deformation) that has been considered responsible for the most severe CNS injury in SBS/NAI.[4,5,10–13] Inertial loading accompanying sudden angular acceleration/deceleration of the head on the neck (as with shaking) produces shear strain deformation and disruption at tissue interfaces, therefore *diffusing* the injury (Fig. 2). The young infant is said to be particularly vulnerable because of weak neck muscles, a relatively large head, and an immature brain. It is the shaking mechanism that is traditionally postulated to result in the triad, including primary traumatic injury (ie, SDH, RH, and DAI), with or without the secondary injury pattern (ie, edema, swelling, hypoxia-ischemia, herniation). Reportedly, such patterns are associated with the most severe and fatal CNS injuries and are readily demonstrated by means of neuroimaging, surgical neuropathology, and postmortem neuropathology.[4,5,10–13]

On a medical forensic basis, it is further stipulated that (1) retinal hemorrhages of a particular pattern are diagnostic of SBS/NAI, (2) such CNS injury on an accidental basis can only be associated with a massive force equivalent to a motor vehicle accident or a fall from a 2-story building, (3) such injury is immediately symptomatic and cannot be followed by a lucid interval, and (4) changing symptoms in a child with previous head injury is caused by newly inflicted injury and not just a *rebleed*. Using this reasoning, the last caretaker is automatically guilty of abusive injury, especially if not witnessed by an independent observer.[4,5,10–13]

The range of acute primary and secondary CNS injury reported to occur with NAI significantly overlaps that of AI.[4,5,10,11] This includes multiple or complex cranial fractures, acute interhemispheric SDH (Fig. 2), acute-hyperacute convexity SDH, multiple contusions, shear injury (DAI, white matter tears), brain swelling, edema, and hypoxia-ischemia (Fig. 2). The range of chronic CNS injury includes chronic SDH, communicating hydrocephalus, atrophy, or encephalomalacia. The combination of acute and chronic findings suggests more than 1 traumatic event. Imaging evidence of CNS injury may occur with or without other clinical findings of trauma (eg, bruising) or other traditionally *higher-specificity* imaging findings associated with violent shaking (eg, metaphyseal, rib, or other typical skeletal injuries).[4,5,10] Therefore, clinical and imaging findings of injury disproportional to the history, and injuries of differing age, have become 2 of the key diagnostic criteria indicating the *probability* of NAI/SBS, particularly when encountered in the premobile, young infant.[4,5,10] Such clinical and imaging findings have traditionally formed the basis from which health professionals, including radiologists, have provided a medical diagnosis and offered expert testimony that such *forensic* findings are *proof* of NAI/SBS.[10]

CONTROVERSY

Fundamental difficulties persist in formulating a *medical* diagnosis or *forensic* determination of NAI/SBS on the basis of a causative event (ie, shaking) that is inferred from clinical, radiological, and/or pathological findings in the often *subjective* context of (1) an unwitnessed event, (2) a *noncredible* history, or (3) an admission or confession.[4,5,10] This problem is further confounded by the lack of consistent and reliable criteria for the diagnosis of NAI/SBS, and that the vast body of literature on child abuse is comprised of

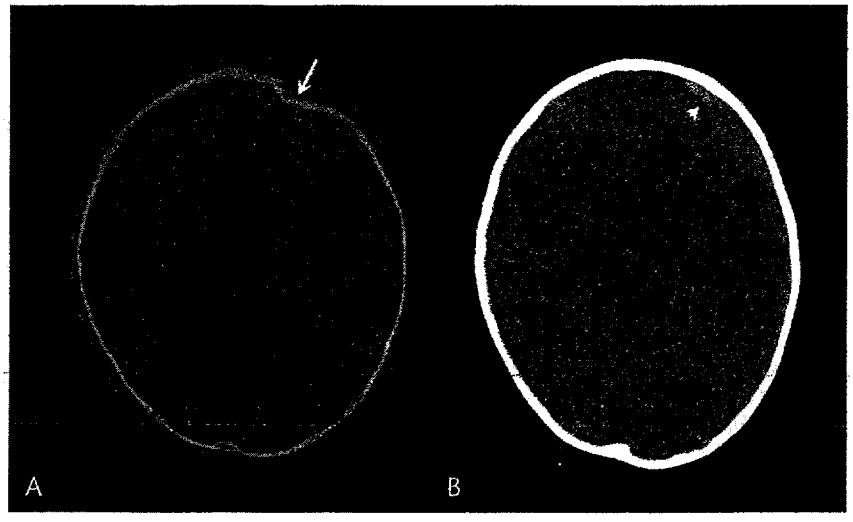

FIGURE 1. Images obtained from a 22-month-old female motor vehicle accident victim with depressed left-side frontal skull fracture (A, arrow), overlying scalp swelling, and a small, high-density epidural hematoma (B, arrowhead).

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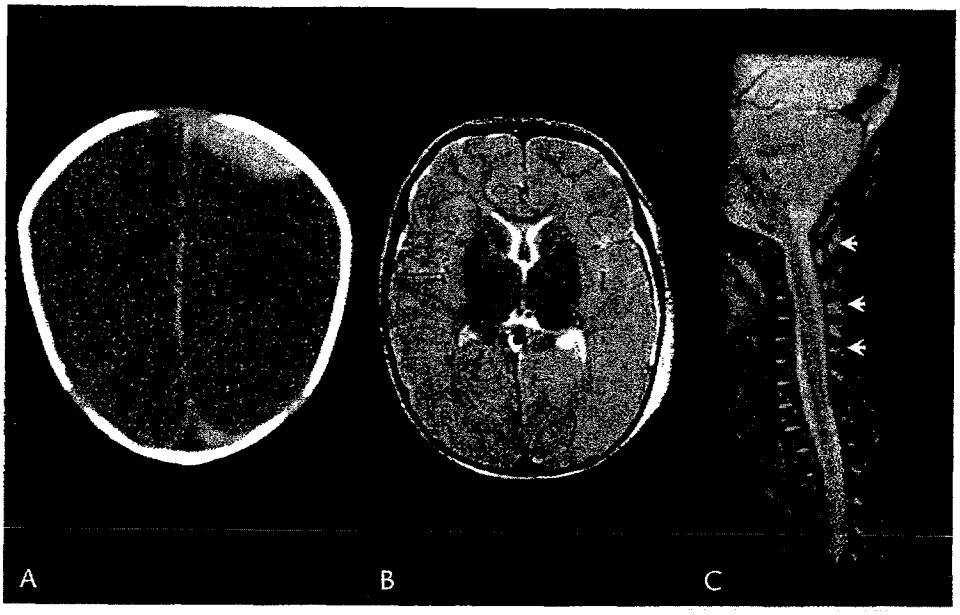

FIGURE 2. Images obtained from a 25-day-old female neonate with history of drop and RH (alleged NAI). A, Axial CT image shows high-density left-side frontal SDH (surgically drained before MRI), bilateral cerebral low densities with decreased gray-white matter differentiation (hypoxia-ischemia?), and interhemispheric high-density hemorrhage. B, Axial T2 MRI scan shows bilateral cerebral cortical and subcortical T2 high intensities plus interhemispheric T2 low intensities. C, Sagittal STIR cervical spine MRI scan shows posterior ligamentous high intensities (arrows) but no definite cord injury (NAI? SCIWORA?).

anecdotal case series, case reports, reviews, opinions, and position papers.[10,14] Furthermore, many reports include cases having impact injury that not only raises doubt regarding the *shaking-only* mechanism but also questions that this injury is always NAI based on a *shaken-impact* mechanism. From the perspective of EBM, QOE ratings for SBS/NAI diagnostic criteria reveal that few published reports merit a rating above class IV (ie, any design where test is not applied in blinded evaluation, or evidence provided by expert opinion alone or in a descriptive case series without controls).[10,14] The inclusion criteria provided in many reports often seem arbitrary, such as *suspected abuse*, *presumed abuse*, *likely abuse*, and *indeterminate*.[15,16] Furthermore, the diagnostic criteria often seem to follow *circular logic* (ie, SBS = SDH + RH [inclusion criteria], therefore SDH + RH = SBS [conclusion]). Such low QOE ratings hardly earn a EBM diagnostic recommendation level of *optional*, much less as a *guideline* or a *standard*.[10,14] This has traditionally been true of the neuroimaging literature, the clinical literature that uses neuroimaging, and the forensic pathology literature.[10,17–44]

The most widely reported attempt of a scientific study to test NAI/SBS used a biomechanical approach, measured stresses from shaking versus impact in a doll model, and correlated those stresses with injury thresholds in subhuman primate experiments established in another study.[45–47] Only stresses associated with impact, whether using an unpadded or padded surface, exceeded the injury thresholds that correlated with the pathological spectrum of concussion, SDH, and DAI. The authors concluded that CNS injury in SBS/NAI in its most severe form is usually not caused by shaking alone. These results obviously contradicted many of the original reports that had relied on the "whiplash" mechanism as causative of the triad.[47–49] These authors also concluded that fatal cases of SBS/NAI, unless occurring in children with predisposing factors (eg, subdural hygroma [SDHG], atrophy, etc), are not likely to result from shaking during play, feeding, and swinging, or from more vigorous shaking by a caretaker for discipline. A number of subsequent studies using various biomechanical, animal, and computer models have failed to convincingly invalidate this study, although many contend that there is no adequate model yet designed to properly test shaking versus impact.[50–61] Some of these reports also indicate that shaking alone cannot result in brain injury (ie, the triad) unless there is concomitant neck, cervical spinal column, or cervical spinal cord injury (Fig. 2).[53,54]

A number of past and more recent reports raise serious doubt that abuse is the cause in all cases of infant CNS injury using traditional SBS/NAI diagnostic criteria.[10,14,16,46,49,62–68] This includes reports of skull fracture or acute SDH from accidental simple falls in young infants, such as those associated with wide extracerebral spaces (eg, benign external hydrocephalus, benign extracerebral collections of infancy, SDHGs),[69–83] and fatal pediatric head injuries caused by witnessed, accidental short-distance falls, including those with a lucid interval and RH.[84–102] Recent neuropathologic studies in alleged SBS cases indicate that (1) the cerebral swelling in young infants is more often caused by diffuse axonal injury of hypoxic-ischemic origin rather than traumatic origin (traumatic origin is more appropriately termed *multifocal traumatic axonal or shear injury*); (2) although Fx, SDH (eg, interhemispheric), and RH are commonly present, the usual cause of death was increased intracranial pressure from brain swelling associated with hypoxia-ischemia; and (3) cervical EDH and focal axonal brain stem, cervical cord, and spinal nerve root injuries were characteristically observed in these infants (presumably caused by shaking, although most had impact findings).[103–109] Such upper cervical cord/brainstem injury may result in apnea/respiratory arrest and be responsible for the hypoxic-ischemic brain injury. Additional neuropathologic series have shown that dural hemorrhages are also observed in nontraumatic fetal, neonatal, and infant cases, and that the common denominator is likely a combination of cerebral

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venous hypertension and congestion, arterial hypertension, brain swelling, and immaturity with vascular fragility further compromised by hypoxia-ischemia or infection.[107–109] Reports of neurosurgical, neuroradiological, and neuropathologic findings in head trauma, as correlated with biomechanical analyses, indicate that SDH and RH occur with rotational deceleration injuries, whether *accidental* (eg, axis or center of rotation internal to the skull, including those resulting from short-distance falls) or *nonaccidental* (ie, axis of rotation external to the skull [eg, at the craniocervical junction or cervical spinal level]).[50–53] There is no scientific basis to date to indicate how much or how little force is necessary to produce traumatic injury to the developing CNS.

Furthermore, the specificity of RH for child abuse and its dating has also been questioned.[4,5,10,16,49,67,68,73,84,110–113] Such hemorrhages have been reported with a variety of conditions, including AT, resuscitation, increased intracranial pressure, increased venous pressure, subarachnoid hemorrhage (SAH), sepsis, coagulopathy, certain metabolic disorders, systemic hypertension, and other conditions. Furthermore, many cases of RH (and SDH) are confounded by the existence of multiple factors or conditions that often have a synergistic influence on the type and the extent of RH. For example, consider the child who has trauma, hypoxia-ischemia, coagulopathy, and has undergone resuscitation.

IMAGING PROTOCOLS

Proper imaging evaluation includes not only computed tomography (CT) and a radiographic or radionuclide skeletal survey but also magnetic resonance imaging (MRI) and, in some cases, serial imaging.[4,10,114–118] Occasionally, ultrasonography (US) may be useful. The imaging protocols should be designed to evaluate not only NAI versus AI but also the nontraumatic mimics. Computed tomography is the primary modality in acute neurological presentations because of its access, speed (particularly using multidetector technology), and ability to demonstrate abnormalities requiring immediate neurosurgical or medical intervention (eg, an expanding hematoma, brain swelling, impending herniation) (Figs. 1, 2).[4,10,114] Nonenhanced head CT with soft tissue and bone algorithms is performed. Facial and spinal (eg, cervical) CT may also be needed, including reformatting. Three-dimensional computed tomographic reconstructions can be important to evaluate fractures versus developmental variants (eg, accessory sutures, fissures, synchondroses). Computed tomographic angiography (CTA) or computed tomographic venography (CTV) may be helpful to evaluate the cause of hemorrhage (eg, vascular malformation, aneurysm) or infarction (eg, dissection, venous thrombosis). Intravenous contrast-enhanced CT or US with Doppler may be used to separate subarachnoid and subdural compartments by identifying bridging veins within the subarachnoid space; however, MRI is usually needed for more definite evaluation. In addition, in the unstable infant, initial and repeat cranial US (eg, transcranial Doppler) at the bedside may assist in evaluating structural abnormalities and monitoring alterations in cerebral blood flow and intracranial pressure.

Magnetic resonance imaging should be conducted as soon as possible because of its sensitivity and specificity regarding pattern of injury and timing parameters.[4,10,114–118] Brain MRI should include 3 planes and at least T1, T2, fluid-attenuated inversion recovery (FLAIR), gradient-recalled echo (GRE) T2*, and diffusion imaging (diffusion-weighted imaging [DWI]/apparent diffusion coefficient [ADC]) (Fig. 3). Gadolinium-enhanced T1 images should probably be used along with MRA and magnetic resonance venography (MRV).

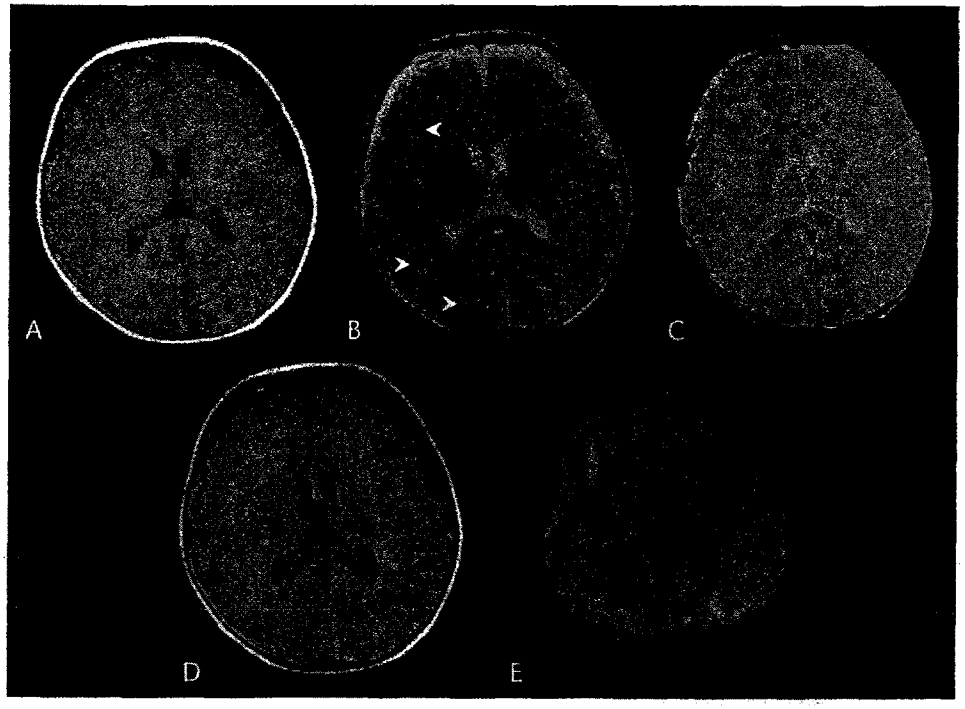

FIGURE 3. Images obtained from an 8-month-old male infant after viral illness, right-side humeral fracture, and RH (alleged NAI). Axial T1 (A), T2 (B), GRE (C), FLAIR (D), and DWI (E) images show bilateral frontal extracerebral CSF-intensity collections with right-side frontal extracerebral hemorrhage that is T1/FLAIR hyperintense and T2/GRE hypointense. Also seen are multifocal cerebral T2/FLAIR hyperintensities (arrowheads) that are DWI hyperintense (shear vs infarction?).

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TABLE 1. Magnetic Resonance Imaging of Intracranial Hemorrhage and Thrombosis*

Stage	Biochemical Form	Site	T1 MRI	T2 MRI
Hyperacute (+ edema) (<24 hours)	Fe II oxyHb	Intact RBCs	Iso-low I	High I
Acute (+ edema) (1–3 days)	Fe II deoxyHb	Intact RBCs	Iso-low I	Low I
Early subacute (+ edema) (3–7 days)	Fe III metHb	Intact RBCs	High I	Low I
Late subacute (− edema) (1–2 weeks)	Fe III metHb	Lysed RBCs (extracellular)	High I	High I
Early chronic (− edema) (>2 weeks)	Fe III transferrin	Extracellular	High I	High I
Chronic (cavity)	Fe III ferritin and hemosiderin	Phagocytosis	Iso-low I	Low I

*Modified from Wolpert and Barnes,[119] Kleinman and Barnes,[4] Bradley,[120] and Zuerrer et al.[121]
RBCs indicates red blood cells; I, intensity; plus sign (+), present; minus sign (−), absent; Hb, hemoglobin; Fe II, ferrous; Fe III, Ferric; Iso, isointense.

The cervical spine should also be imaged, along with other levels when indicated, and especially by using short TI inversion recovery (STIR) (Fig. 2). T1- and T2-weighted imaging techniques are necessary for characterizing the nature and timing (whether hyperacute, acute, subacute, or chronic) of hemorrhages and other collections by using established criteria (Table 1). Gradient-recalled echo or other susceptibility-weighted (T2*) techniques is most sensitive for detecting hemorrhage or thromboses that are often not identified on other sequences. However, GRE cannot be used for timing alone because it shows most hemorrhages (new and old) as hypointense (eg, deoxyhemoglobin, intracellular methemoglobin, hemosiderin).[4,10,114] The FLAIR sequence suppresses cerebrospinal fluid (CSF) intensity and allows for a better assessment of brain abnormalities, especially when adjacent to a CSF space or collection. FLAIR is also sensitive (but nonspecific) for subarachnoid space abnormalities, which appear as high intensity (eg, hemorrhage, exudate, inflammatory or neoplastic leptomeningeal infiltration, occlusive vascular slow flow, and hyperoxygenation during sedation or anesthesia). DWI plus ADC can be quickly obtained to show hypoxia-ischemia or vascular occlusive ischemia. Magnetic resonance spectroscopy (MRS) may show a lactate peak. It must be remembered, however, that restricted or reduced diffusion may be observed in other processes, including encephalitis, seizures, or metabolic disorders, and with suppurative collections and some tumors.[4,10,114] Gadolinium-enhanced sequences and MRS can be used to evaluate these other processes. In addition, MRA and MRV are important to evaluate arterial occlusive disease (eg, dissection) or venous thrombosis. The source images should be viewed along with the reprojected images. In some cases of partial occlusion/thrombosis, the abnormality may be more conspicuous on CTA/CTV, especially in infants. For evaluating arterial dissection by means of MRI, an axial fat-suppressed T1 sequence from the aortic arch to the circle of Willis may detect T1-hyperintense hemorrhage or thrombosis (ie, methemoglobin) within the false lumen, especially if the process is in the subacute phase.

INJURY EVALUATION

The range of CNS injury in childhood trauma, whether AI or NAI, often demonstrated by imaging may be categorized according to being primary or secondary (as previously described) and according to specific anatomical involvement, including scalp, cranial, intracranial, vascular, spinal, and head and neck.[2,4,5,10] A thorough analysis of the injury requires a systematic breakdown into injury components for both pattern of injury and timing parameters.

SCALP INJURY

Scalp injuries include hemorrhage, edema, or laceration and may be localized to any layer (SCALP [skin, subcutaneous,

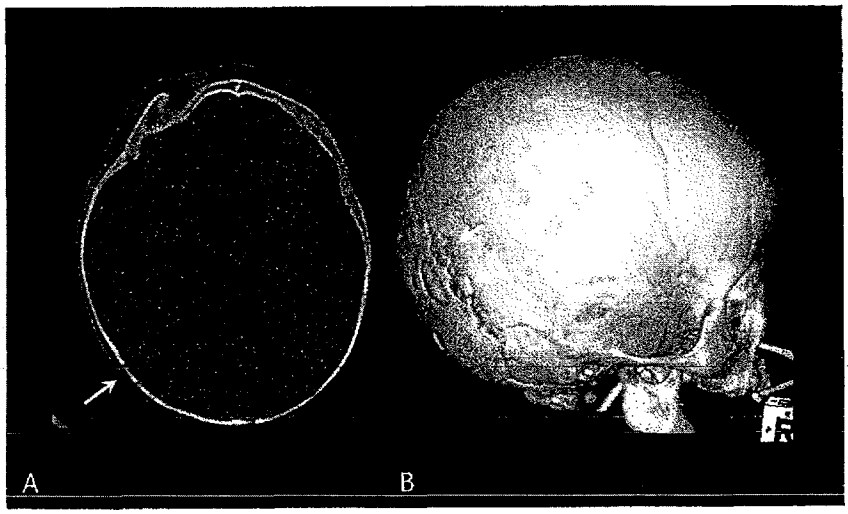

FIGURE 4. Images obtained from a 10-month-old male infant with intrasutural (wormian) bones versus fractures. A, CT image shows right-side parietal cranial defects (arrow). B, Three-dimensional computed tomographic surface reconstruction confirms intrasutural bones (arrows).

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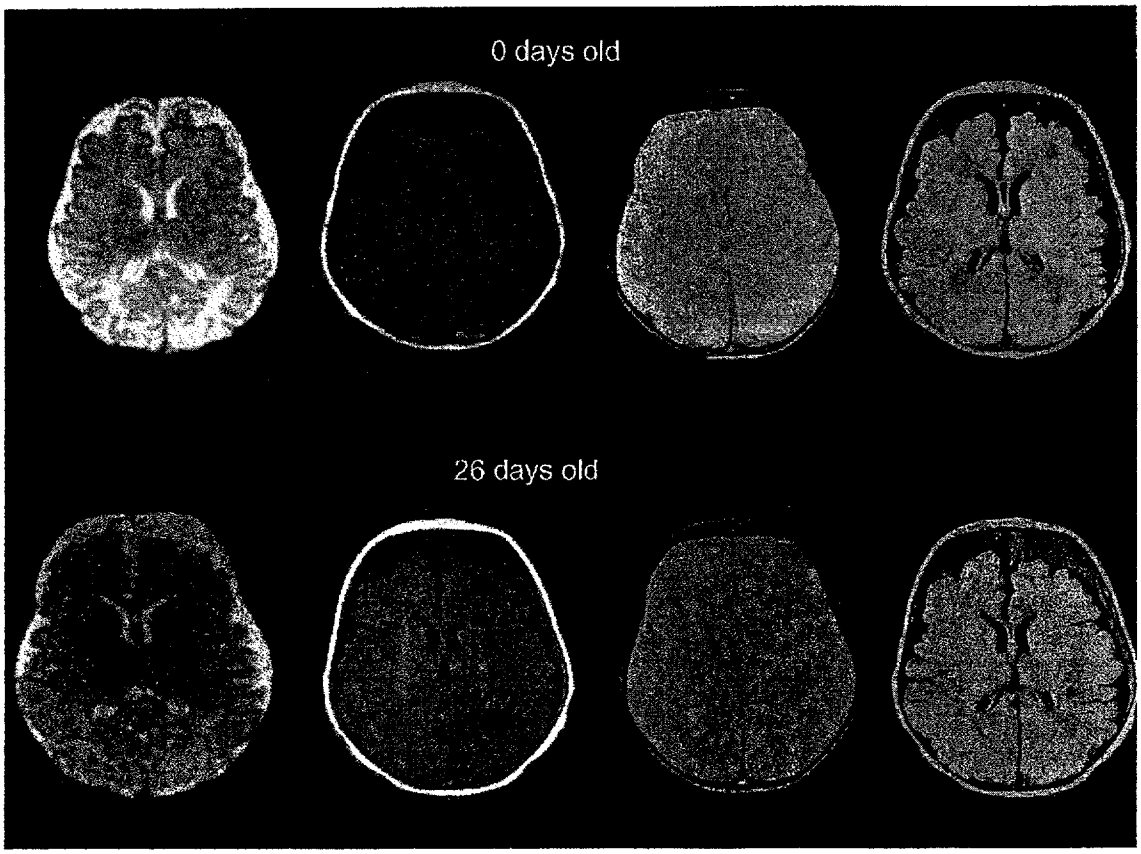

FIGURE 5. Images obtained from an infant with benign extracerebral collections of infancy and spontaneous subdural hemorrhage. Axial T2, T1, GRE, and FLAIR images (left to right) show CSF-intensity frontal subarachnoid collections at birth (top row). At 26 days postnatal age (bottom row), superimposed subdural collections that don't conform to CSF signal are present (courtesy of Veronica J. Rooks, MD, Tripler Army Medical Center, Honolulu HI).

galea *a*poneurotica, *l*oose or subga*l*eal space, *p*eriosteum]).[2,4,5,10] Although CT or MRI may not precisely resolve scalp layers, the site of a collection may be inferred by means of morphological findings (Fig. 1). Subperiosteal collections (eg, cephalohematoma) are usually confined by the sutures. Subcutaneous or subgaleal collections are not as contained, may be more extensive, and can contribute to circulatory compromise. Scalp injuries are difficult to precisely time on imaging studies, unless serial examinations are available; in addition, timing depends on the nature and the number of traumatic events or other factors (eg, circulatory compromise). Unless there is direct vascular injury that results in an acute hematoma, collections or edema may not be identified on early imaging. Scalp injuries may become evident several hours later or on the next day. Nonvisualization of scalp or skull abnormalities on imaging should not be interpreted as absence of impact injury.

SKULL INJURY

The spectrum of cranial injury includes Fx*s* and suture splitting.[2,4,5,10] Fractures may be simple (eg, single, linear, nondisplaced) or complex (eg, bilateral, multiple, diastatic, depressed, or growing [ie, leptomeningeal cyst]). Localized suture splitting may indicate traumatic diastasis where widening occurs as a part of Fx extension. Diffuse or multiple suture widening may indicate increased intracranial pressure from any cause to include edema, expanding collection, or hydrocephalus. Evaluating the skull in neonates, infants, and young children is challenging because Fx may not be distinguished from sutures, synchondroses, or their normal variations. This is particularly difficult in the parietooccipital region and skull base where accessory sutures, fissures, and synchondroses are common. The significance of this distinction is important because the reporting of a skull Fx is *evidence* of trauma (Fig. 1). In such cases, 3-dimensional computed tomography with surface reconstructions may provide clarification (Fig. 4). In general, the morphology of an Fx does not differentiate NAI from AI. Complex or bilateral skull Fx in this age group can arise from a single event under circumstances other than a 2-story fall or a motor vehicle accident. Such examples include a fall or a drop with impact to the skull vertex, impact against more than 1 surface (eg, table, wall, or floor), fall or drop downstairs, and an adult or older child falling with or onto a smaller child. Skull Fx*s* are also difficult to time by using plain films and CT because of the lack of periosteal reaction during healing. A simple skull Fx in an infant may require 6 months for complete healing. In an older child and adult, this may take up to a

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year.[2,4,5,10] Intracranial air densities (ie, pneumocephalus) may be related to fracture involving the paranasal sinuses or otomastoid structures, caused by penetrating trauma (eg, open skull fracture), arise from CSF access (eg, lumbar puncture) or vascular access (eg, indwelling catheter), or may be associated with gas-forming infections.

EXTRACEREBRAL COLLECTIONS

The range of intracranial injury includes abnormal fluid collections and brain injury.[2,4,5,10] Abnormal collections may be subarachnoid, intraventricular, subdural, or epidural. These may contain hemorrhage of any age (eg, hyperacute, acute, subacute, chronic, combined), cerebrospinal fluid (CSF [eg, hygroma, hydrocephalus]), protein, exudate, or any combination of elements. On imaging, it may be impossible to specifically define the components or age of a collection (eg, SDHG vs chronic SDH). Subarachnoid and subdural collections may be localized or extensive and occur near the convexities, interhemispheric (along the falx), and along the tentorium. Epidural hemorrhage, whether arterial or venous in origin, tends to be more localized (limited by the periosteal layer of the dura mater along the inner calvarial table) and can cross midline (Fig. 1). Epidural (intradural) hemorrhage may split the leaves of dura and collect within the tentorium or falx. Epidural collections usually appear lentiform. Subdural collections tend to be crescentic and follow the contour of the adjacent cerebrum or cerebellum (Fig. 3). Subarachnoid collections may be less well defined (unless loculated) and extend into cisterns, fissures, or sulci. Occasionally, a collection cannot be determined to be specifically subarachnoid, subdural, or epidural because collections in multiple spaces may be present, owing to membrane layer disruption (Fig. 2). Intraventricular hemorrhage is a rare but reported finding in trauma. It may also be an indicator of associated hypoxia-ischemia, coagulopathy, or venous thrombosis.

Prominent subarachnoid CSF spaces may normally be present in infants (aka benign extracerebral collections [BECC], benign extracerebral subarachnoid spaces, benign external hydrocephalus).[10,79–83,114] These should be of the same density/intensity as CSF on CT and MRI (Fig. 5). This condition predisposes infants to SDH, which may be spontaneous or associated with trauma of any type (Fig. 5). A hemorrhagic collection may continually change or evolve with regard to size, extent, location, and density/intensity characteristics. Cases of rapid resolution and redistribution of acute SDH for a few hours to 1 to 2 days have been reported.[117,122] A tear in the arachnoid may allow SDH washout into the subarachnoid space or CSF dilution of the subdural space. An SDH may also redistribute within the subdural space as a gravity-dependent process (eg, a convexity SDH migrating to the peritentorial and posterior interhemispheric regions)[114,117] (Fig. 6). Subdural hemorrhage migration may lead to misinterpretation of a new hemorrhage. The distribution or migration of the sediment portion of a hemorrhage with blood levels (ie, hematocrit effect) may

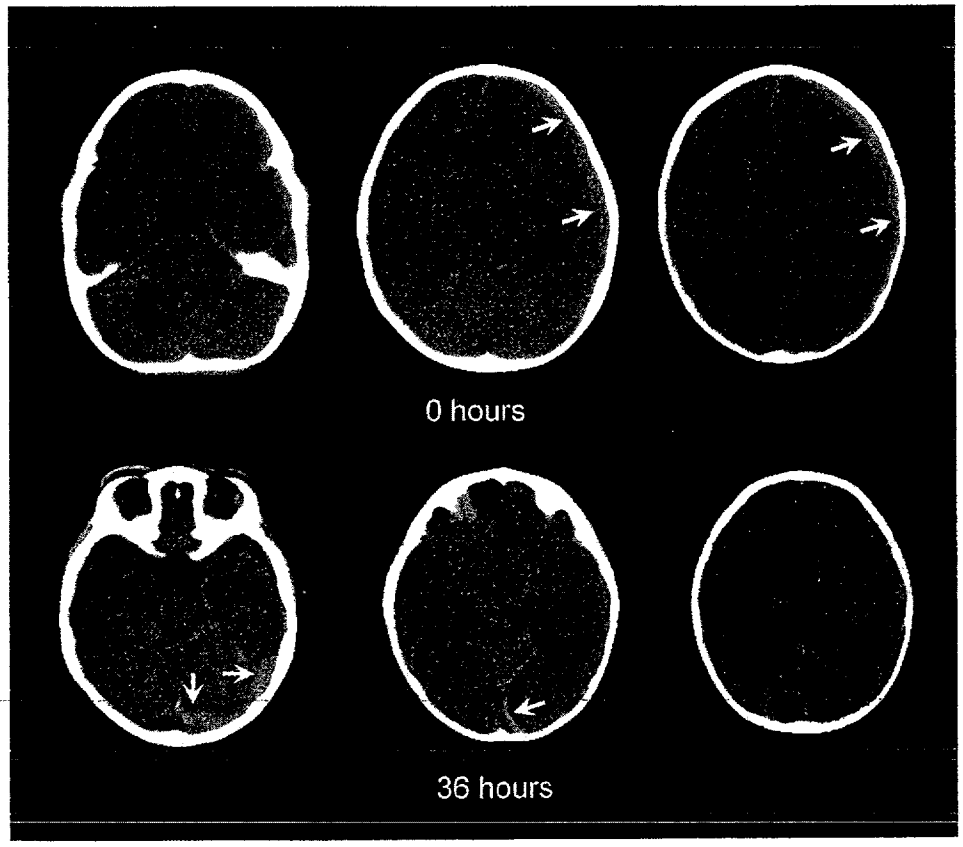

FIGURE 6. Images obtained from a 9-month-old female infant who had accidental trauma from left-side frontal impact. Computed tomographic images at presentation (top row) show left-side frontotemporal-convexity high-density subdural hemorrhage (arrows). Computed tomographic images obtained after 36 hours in the hospital (bottom row) show redistribution of the high-density hemorrhage to the peritentorial region and posterior interhemispheric fissure (arrows).

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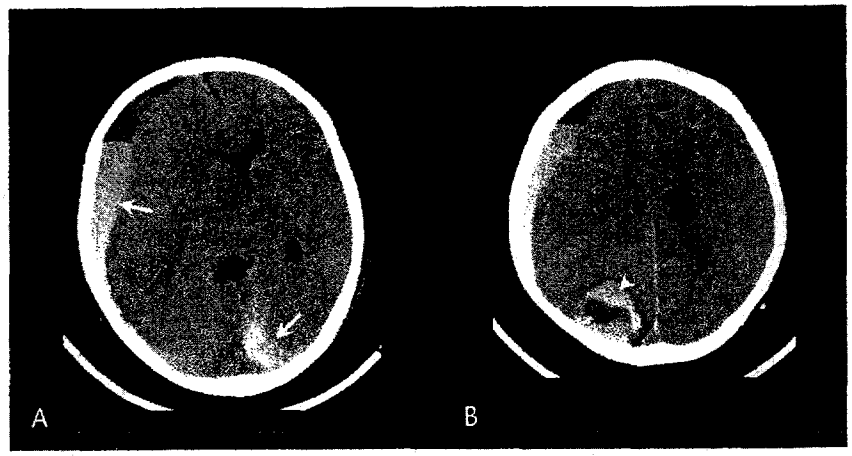

FIGURE 7. Images obtained from a 2-year-old boy with congenital heart disease and ECMO. Axial computed tomographic images show bilateral subdural hematomas (A, arrows) and right-side parietal intracerebral hematoma (B, arrowhead) with low-density over high-density fluid levels.

cause further confusion because the density/intensity differences between the sediment and supernatant may be misinterpreted as hemorrhages (and trauma) of differing age and location (Figs. 7, 8).[117] In addition, more recent reports further substantiate that (1) the interhemispheric SDH may be observed in AI and, therefore, is not specific for NAI; (2) mixed-density SDH also occurs in AI; (3) SDH may occur in BECC either spontaneously or as a result of minor trauma (ie, AI); and (4) rehemorrhage within SDH may occur spontaneously or with minor AI.[10,82,114–118]

BRAIN INJURY

Traumatic brain injury includes contusion, shear injury, hemorrhage, and edema.[2,4,5,10] Contusions represent focal or multifocal impact injury, are usually hemorrhagic, and typically occur in cortical gray matter along brain surfaces that impact skull bone or dura mater (eg, falx, tentorium). The inner table of the immature, infant skull is not as *rough* as in older children and adults. Therefore, sliding contusions of the frontal or temporal lobes along the floor of the anterior or middle cranial fossa, respectively, occur less often. Infant contusions more commonly occur at the primary site of impact (ie, coup injury) or at a secondary, "recoil" site opposite the primary impact (ie, contracoup injury). Shear injury (ie, traumatic axonal injury, white matter tear) is also focal or multifocal and typically occurs at deep gray matter–white matter junctions, along the corpus callosum, and within the brain stem (Fig. 3). They are more often nonhemorrhagic but may become hemorrhagic. In severe cases, shear injuries may appear as gross tears. This type of injury has been previously referred to as *diffuse axonal injury* or DAI. It is more properly termed *multifocal or traumatic axonal injury* because diffuse axonal injury is more characteristic of hypoxic-ischemic injury (Fig. 2).[104–109]

Edema or swelling may be traumatic, hyperemic, hypoxic-ischemic, or related to other factors (eg, seizures, metabolic).[2,4,5,10] Traumatic edema is related to direct traumatic effects such as contusion, shear, or the result of a vascular injury (eg, dissection, herniation) (Figs. 2, 3). Malignant brain edema, a term used for severe cerebral swelling leading to rapid deterioration, may also occur in children with head trauma. The edema may be related to cerebrovascular congestion (ie, hyperemia) as a vasoreactive rather than an autoregulatory phenomenon. There may be rapid or delayed onset.[84–96] Predisposing factors are not well established but likely include a genetic basis. Global hypoxia (eg, apnea, respiratory failure) or ischemia (eg, cardiovascular failure or dissection) is likely a major cause of or contributor to brain edema in the child with head trauma (Fig. 2). Other contributors to edema or swelling include such complicating factors as seizures (eg, status epilepticus), fluid-electrolyte imbalance, other systemic or metabolic derangements (eg, hypoglycemia, hyperglycemia, hyperthermia), or hydrocephalus. The type (eg, cytotoxic, vasogenic, hydrostatic) and pattern of edema tend to conform to the nature and distribution of the causative insult. Traumatic edema is often focal or multifocal (eg, in areas of contusion, shear, or hemorrhage) (Fig. 3). Hyperemic edema is often diffuse and may appear early as accentuated gray-white matter differentiation on CT, then progressing to loss of differentiation (Fig. 2). Hypoxic-ischemic injury, depending on its severity and duration, may have a diffuse appearance acutely with decreased gray-white matter differentiation throughout the cerebrum on CT (eg, white cerebellum sign) and then evolve to a more specific pattern on CT or MRI (eg, border zone or watershed, basal ganglia/thalamic, cerebral white matter necrosis, reversal sign) (Fig. 2).[10,114,123–126] The subacute to chronic sequelae of traumatic brain injury include hydrocephalus, atrophy, encephalomalacia, gliosis, mineralization, and chronic extracerebral collections.

VASCULAR INJURY

Arterial trauma may result in dissection or pseudoaneurysm.[2,4,5,10,123, 127] The vascular injury may be the result of penetrating or nonpenetrating trauma, may be spontaneous, or caused by existing disease (eg, arteriopathy). Internal carotid artery dissection typically involves the cervical or supraclinoid segments. Vertebrobasilar dissection most commonly involves the distal cervical portion of the vertebral artery at the C1-C2 level. Intracranial or multiple dissections may rarely occur. Dissection may result in stenotic, thrombotic, or embolic infarction. Pseudoaneurysms may be associated with hemorrhage. The vascular injury may be initially detected by means of CT and CTA (Fig. 9) or of MRI (eg, DWI, axial fat-suppressed T1 sections of the neck and skull base) with MRA. Catheter

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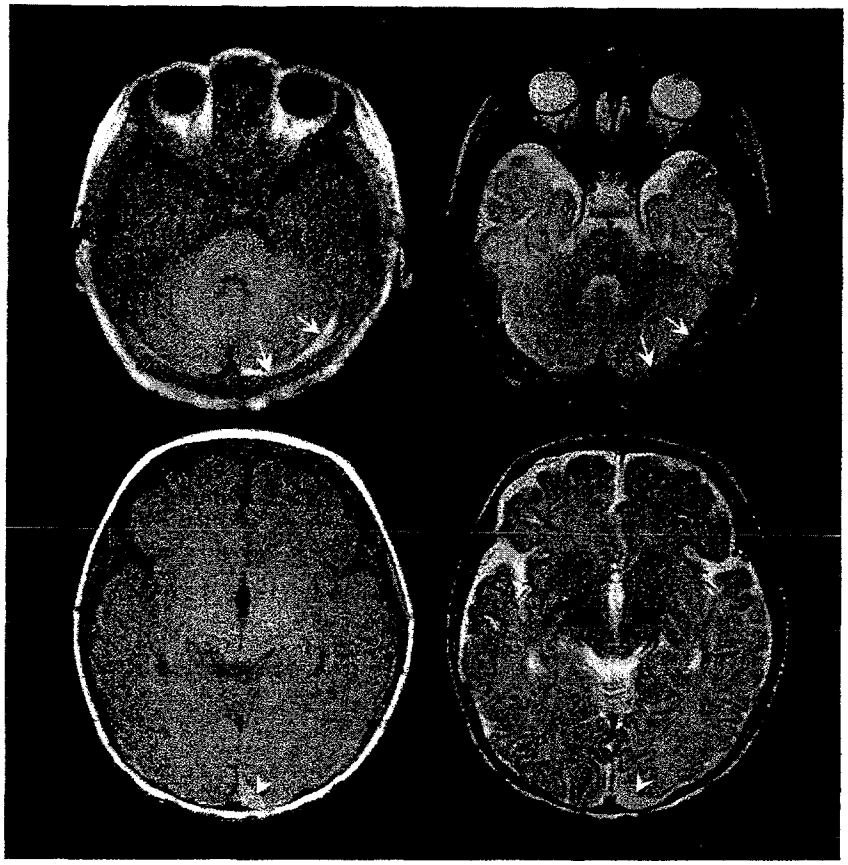

FIGURE 8. Images obtained from a 2-month-old female infant with left-side peritentorial and posterior interhemispheric subdural hemorrhage. Axial MRI images show T1-hyperintense and T2-hypointense sediment along the tentorium (top row, arrows) with T1- and T2-isohyperintense supernatant above (bottom row, arrowheads).

angiography may be necessary for definitive evaluation. Arterial occlusive infarction also occurs with the various types of herniation, in which relatively specific distributions are observed. Dural sinus and venous thrombosis may also occur with trauma (eg, adjacent to fracture, associated or predisposing coagulopathy) or as a mimic of NAI (eg, infection, coagulopathy).[128] Computed tomography may show hyperdensity within the venous system, a focal venous enlargement with associated subarachnoid or subdural hemorrhage, or infarction that is often hemorrhagic. A more definitive diagnosis may be made by means of CTV or of MRI and MRV.

SPINAL INJURY

The spectrum of spinal injury in NAI significantly overlaps that of AI.[2,4,5,10,123] This spectrum differs with age (degree of spinal development) and includes either single or

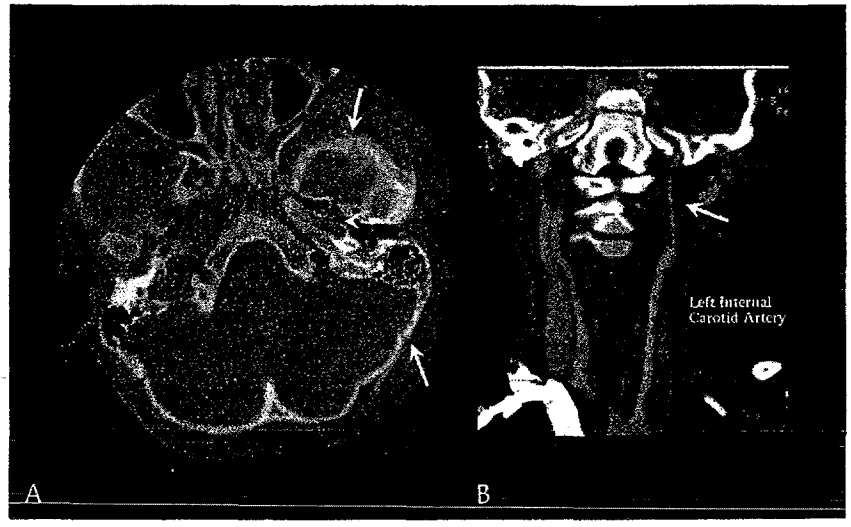

FIGURE 9. Images obtained from a 5-year-old boy. A, Computed tomographic image shows left-side skull base fractures involving left-side occiput, petrous bone, and sphenoid wing (arrows). Air densities are seen within the carotid canal (arrowhead). B, Computed tomography angiogram shows left-side cervical internal carotid arterial dissection with marked luminal narrowing (arrow).

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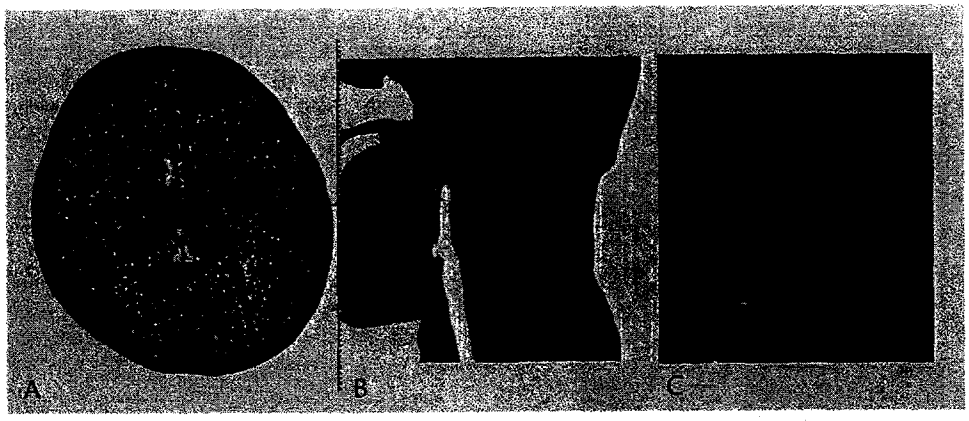

FIGURE 10. Images obtained from a 22-month-old boy with SCIWORA (caused by backward fall and parietal head impact) and hypoxic-ischemic injury and RHs. A, Axial brain CT image shows (1) bilateral cerebral low densities with decreased gray-white matter differentiation (edema) and (2) small high-density asymmetrical cerebral, extracerebral, and posterior interhemispheric hemorrhages. B, Sagittal reformatted cervical spinal computed tomographic image shows no spinal column abnormality (MRI not performed). C, Postmortem midsagittal section shows cervicomedullary disruption (circle). Diffuse hypoxic-ischemic axonal brain injury was also confirmed.

multiple lesions involving the cervical, thoracic, lumbar, or sacral level. The mechanisms of injury include hyperflexion, hyperextension, axial loading or rotation, and distraction. The range of spinal column and paraspinal injury includes vertebral or neural arch fractures, bony fragment or disk displacement, dislocations, instability, and paraspinal ligamentous, muscular, or vascular injury. Such injuries may not be apparent on plain films (eg, spinal cord injury without radiographic abnormality [SCIWORA]) and require additional CT plus MRI for complete evaluation.[129–131] Magnetic resonance imaging is particularly important for evaluating ligamentous injury and intraspinal injury. The range of intraspinal injury includes displaced bone or disk fragments and hematomas (eg, epidural) with spinal cord or nerve root compression. There may be edema, contusion, hemorrhage, transection of the spinal cord, or avulsion of 1 or more nerve roots. Computed tomographic angiography or MRA may be needed to evaluate vascular injury (eg, dissection). Cervical spinal cord injury may be associated with head injury or may be the unsuspected cause of respiratory failure and hypoxic-ischemic brain injury (eg, SCIWORA) (Fig. 10).[129–131] This should be evaluated by means of MRI in all such cases, whether AI or NAI. In addition, one must be aware of predisposing conditions that may result in major neurological deficits associated with *minor* head and neck trauma mechanisms (eg, craniocervical anomaly with instability Fig. 11; Chiari I malformation Fig. 12).

IMAGING ANALYSIS—COMPUTED TOMOGRAPHY

Regarding the initial computed tomographic examination, the findings are often nonspecific with regard to pattern of injury and timing and require a differential diagnosis (DDX). To properly analyze such a case from an imaging perspective, each injury component must be addressed separately, and then collectively, and then correlated with clinical and other data.[4,10,114] The major findings are often (1) extracerebral and cerebral high densities, (2) extracerebral isohypodensities, (3) cerebral low densities, with or without (4) scalp or skull abnormalities. In general, the DDX may include trauma (AI vs NAI), hypoxia-ischemia, ischemic injury (arterial vs venous occlusive disease), seizure edema, infectious or postinfectious conditions, coagulopathy, fluid-electrolyte derangement, metabolic or connective tissue disorder, and multifactorial.

Extracerebral high densities are often seen posteriorly along the tentorium, falx, interhemispheric fissure, and dural

FIGURE 11. Image obtained from an 8-year-old girl with Down syndrome and minor trauma with quadriparesis. Sagittal T2 MRI scan shows hypoplastic dens, os odontoideum (anterior arrow), and anterior atlantoaxial instability (confirmed by means of CT) with cervicomedullary compression and high-intensity edema (posterior arrows).

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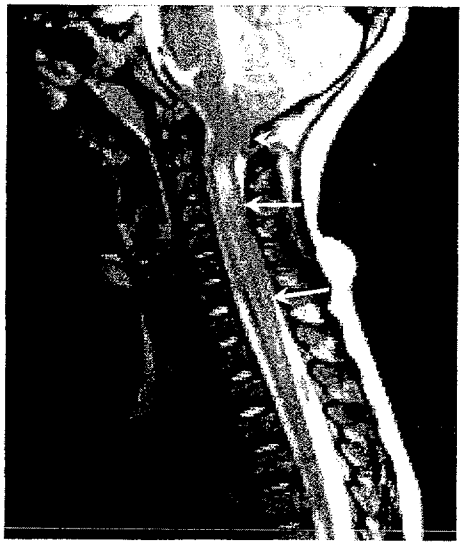

FIGURE 12. Image obtained from a 3-year-old boy with Chiari I malformation, minor trauma, and subsequent quadriparesis. Sagittal T2 MRI scan shows cerebellar tonsils extending into the upper cervical canal (upper arrowhead) and diffuse high-intensity edema of the cervical spinal cord (lower arrows). No abnormality was present on plain films or CT (SCIWORA).

venous sinuses that may vary in laterality and symmetry (Figs. 2, 6, 7, 10, 13–16). These and other extracerebral high densities may be laminar, linear, nodular, or punctate. Using published criteria and timing parameters (discussed in the succeeding sections), these represent either acute to subacute hemorrhages (subarachnoid, subdural) or thromboses (eg, venous).[4,10,114–118] For apparent intracerebral high densities, it may be difficult to differentiate cerebral from SAHs (including those within the perivascular spaces) from vascular thromboses (eg, cortical, subependymal, or medullary venous thromboses). Computed tomography may not be able to distinguish focal or multifocal cerebral high densities as hemorrhagic contusion, hemorrhagic shear, or hemorrhagic infarction (Figs. 13, 16, 18). Extracerebral isohypodensities may represent subarachnoid spaces (eg, BECC), SDHG, hyperacute SDH, or chronic SDH (Figs. 14, 17). According to the literature, the timing for any of the mentioned findings is as follows: (1) hemorrhage or thromboses that are high density (ie, clotted) on CT (ie, acute to subacute) have a wide timing range of 3 hours to 7 to 10 days (Figs. 1, 2, 6, 7, 10, 13–18), (2) hemorrhage that is isohypodense on CT (ie, nonclotted) may be hyperacute (timing, <3 hours) or chronic (timing, >10 days) (Figs. 14, 17), (3) the low density may also represent preexisting wide, CSF-containing subarachnoid spaces (eg, BECC) or SDHG (ie, CSF containing) that may be acute or chronic (Figs. 14, 17), (4) blood levels are unusual in the subacute unless there is coagulopathy (Fig. 7), (5) CT cannot distinguish acute hemorrhage from rehemorrhage on existing chronic collections (BECC or chronic SDHG) (Fig. 17), and (6) the interhemispheric SDH is no longer considered characteristic of NAI (Figs. 2, 6, 7, 13–16).[4,10,114–118]

Cerebral low densities may vary in bilaterality and symmetry and be associated with decreased gray-white matter differentiation or mass effect (Figs. 2, 10, 17). In general, this indicates edema/swelling, the timing of which depends on causation. If related to trauma, such edema/swelling may represent primary injury or secondary injury and be acute-hyperacute (eg, timing of few hours) or delayed (eg, timing of several hours to a few days), including association with lucid interval and short falls.[4,10,114,123–126] Bilateral diffuse edema is most commonly observed in hypoxia-ischemia but may also be observed in other diffuse processes (eg, fluid-electrolyte imbalance, status epilepticus, encephalitis, etc). Focal or multifocal edema may be observed in contusion (eg, gray matter), shear (eg, white matter), infarction (gray or white matter), encephalitis, or demyelination (eg, acute disseminated encephalomyelitis).

Cranial defects may represent Fx, and their timing range is very broad (eg, hours to months old) (Fig. 1).[4,10,114] Furthermore, Fx morphology (eg, multiple, growing) does not reliably distinguish accidental from nonaccidental causation. Scalp collections (hemorrhage, edema, blood level) are also nonspecific with regard to causation and timing (Fig. 1).[4,10,114] If caused by trauma, the timing range is also rather broad (eg, hours to days old). Sutural widening may indicate diastatic Fx

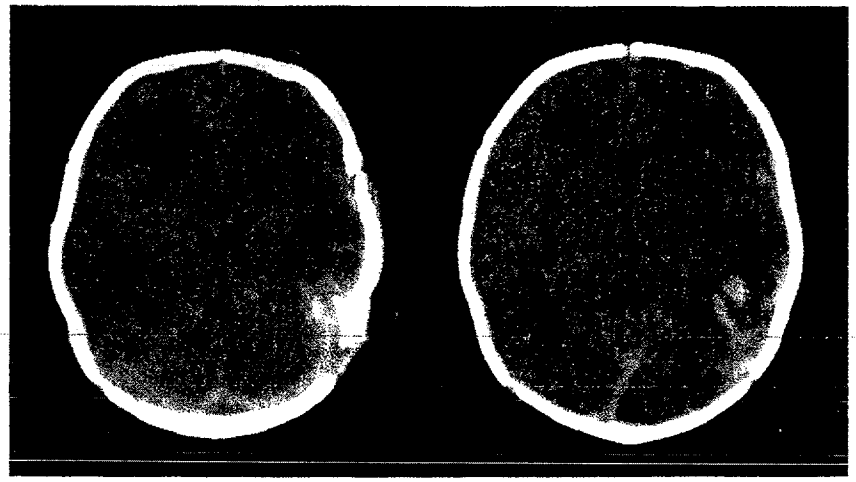

FIGURE 13. Images obtained from a 1-day-old female infant delivered by means of spontaneous vaginal delivery and with subsequent apneic episodes. Computed tomography demonstrates left-side temporal cerebral and extracerebral high-density hemorrhage (or thromboses); high-density hemorrhage is also demonstrated along the interhemispheric fissure, tentorium, and dural venous sinuses. The results of coagulopathy test and sepsis workup were negative (final diagnosis, birth trauma?).

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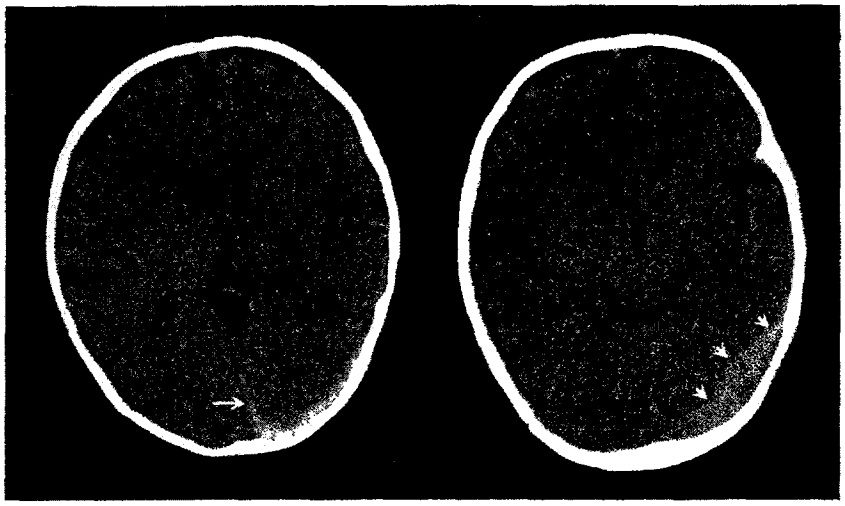

FIGURE 14. Images obtained from a 4-month-old male infant with 2-week viral illness who progressed to septic shock (*Staphylococcus aureus*), endocarditis, severe mitral regurgitation, and coagulopathy. Noncontrast axial CT images show high-density extracerebral hemorrhages (and/or thromboses) along the left-side tentorium, dural venous sinuses, falx, and interhemispheric fissure (arrows). In this case, the bifrontal low-density extracerebral spaces likely represent slightly prominent infantile subarachnoid spaces (BECC?) or underdevelopment, rather than chronic SDH or subdural hygroma.

or increased intracranial pressure. Accessory sutures or synchondroses and developmental fissures may mimic Fx. Intrasutural bones (eg, wormian) may be associated with a skeletal dysplasia or metabolic disorder (Fig. 4).

Subsequent or follow-up computed tomographic examinations may show surgical changes (eg, postevacuation, ventricular catheter, pressure-monitoring device), evolving, redistributing, or recurrent/new hemorrhages, and evolving cerebral densities (edema/swelling). Subsequent CT examinations during the weeks or months may show evolution to permanent cerebral tissue loss (ie, atrophy, encephalomalacia).

IMAGE ANALYSIS—MAGNETIC RESONANCE IMAGING

On an imaging basis, only MRI may provide more precise information regarding pattern of injury and timing, particularly with regard to (1) hemorrhage versus thromboses, and (2) brain injury. The MRI should be performed as soon as feasible, and the findings be compared with the findings from the earlier CT. As a result, MRI has become the standard for such evaluation in these matters.[4,10,114–117,121,123–126]

Hemorrhages and Thromboses

Using published MRI guidelines (Table 1), in general, the evolutionary timing for hemorrhages or thromboses (eg, venous) are as follows: (1) hyperacute phase (timing, <12 hours): T1 isohypointense, T2 hyperintense; (2) acute phase (timing, 1–3 days): T1 isohypointense, T2 hypointense; (3) early subacute phase (timing, 3–7 days): T1 hyperintense, T2 hypointense; (4) late subacute phase (timing, 7–14 days): T1 hyperintense, T2 hyperintense; (5) early chronic phase (timing, >14 days): T1 hyperintense, T2 hyperintense; (6) late chronic phase (timing, >1 to 3 months): T1 isohypointense, T2 hypointense.[4,10,114–117,121,123–124] Mixed intensity collections are problematic regarding timing. Matching the MRI findings with the computed tomographic findings may help, along with follow-up MRI. Blood levels may indicate subacute hemorrhage versus coagulopathy. The timing guidelines are better applied to the sediment than to the supernatant. In addition, a single MRI may not reliably differentiate T1-hypointense/T2-hyperintense collections as representing CSF collections (eg, BECC, acute SDHG) versus hyperacute SDH versus chronic collections (SDH, SDHG). Gradient-recalled echo hypointensities are iron sensitive but do not assist with timing unless matched with

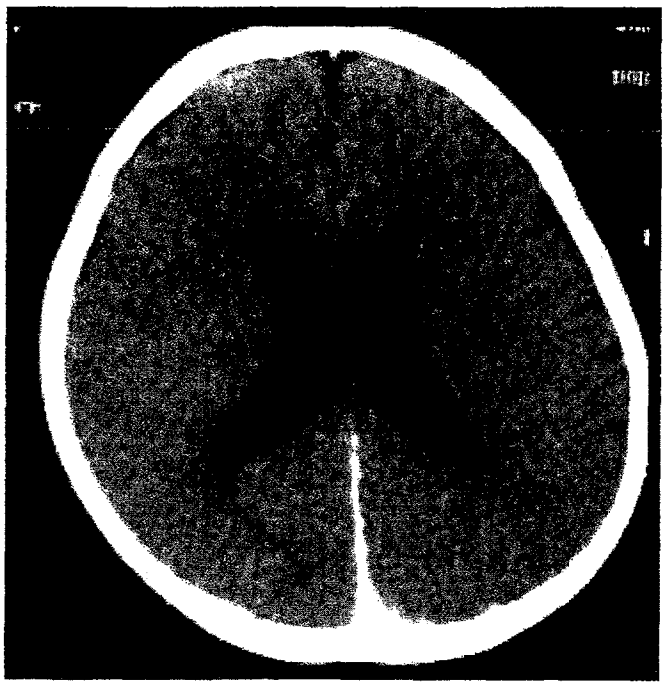

FIGURE 15. Image obtained from a 23-month-old girl who had recent viral gastrointestinal illness, ALTE, RHs, then brain death. Computed tomographic image shows posterior interhemispheric high densities at the level of portions of the inferior sagittal, straight, and superior sagittal sinuses, plus poor cerebral gray-white matter differentiation and moderate ventriculomegaly. Autopsy showed extensive dural and cerebral venous sinus thrombosis with extensive hypoxic-ischemic diffuse axonal brain injury.

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FIGURE 16. Images obtained from a 19-month-old boy who had 1 week of febrile illness (treated with antibiotics), followed by ALTE with RHs. A, Computed tomographic image shows high-density hemorrhages (or thromboses) along the right tentorium and dural venous sinuses. B, Magnetic resonance imaging with MRV shows irregular flow gaps with incomplete opacification of the right-side internal jugular vein and sigmoid sinus. Other flow gaps were demonstrated within the superior sagittal and straight sinuses, along with multiple venous collaterals (diagnosis, DVST).

T1, T2, and computed tomographic densities. Gradient-recalled echo and other magnetic susceptibility sequences are also sensitive to venous thromboses (eg, cortical, medullary, subependymal) that are not detected by means of MRV.

Brain Injury

With regard to brain injury, MRI may distinguish hypoxic-ischemic injury (diffuse relatively symmetrical DWI/ADC restricted diffusion with or without matching T1/T2 abnormalities) from shear and contusional injury (focal/multifocal restricted diffusion, GRE hypointensities, with T2/FLAIR edema). Shear and contusional injury, however, may not be reliably differentiated from focal/multifocal ischemic or hemorrhagic infarction (eg, dissection, vasculitis, venous, embolic) without supportive MRA, CTA, MRV, or angiography.[4,10,114,123–125] In addition, similar cortical or subcortical intensity abnormalities (including restricted diffusion) may also be observed in encephalitis, seizures, and metabolic disorders. Using published MRI criteria and parameters,[114,123–126] in general, the evolutionary timing for ischemic injury is as follows: (1) hyperacute phase (timing, <1 day): DWI hyperintense, ADC hypointense; MRS result, lactate peak; (2) early acute phase (timing, 1–2 days): additional T2 hyperintensity; (3) late acute phase (timing, 2–4 days): additional T1 hyperintensity; (4) early subacute phase (timing, 6–7 days): additional T2 hypointensity; (5) late subacute phase (timing, 7–14 days): additional DWI isohypointense, ADC isohyperintense; (6) chronic phase (timing, >14 to 21 days): additional atrophy. If related to trauma, focal/multifocal ischemic findings may be caused by arterial injury (eg, dissection), venous injury (eg, tear, thrombosis), arterial spasm (as with any cause of hemorrhage), herniation, or edema with secondary perfusion deficit or seizures (eg, status epilepticus). Hypoxia-ischemic brain injury caused by apnea/respiratory arrest may occur with head trauma or with neck/cervical spine/cord injuries (eg, SCIWORA), whether AI or NAI.[114,123,129–131] It may also occur with any nontraumatic cause (eg, choking, paroxysmal coughing, aspiration).[132] In addition to the diffuse brain injury, there may be associated subarachnoid and subdural hemorrhage without mass effect.[104–109]

CONDITIONS MIMICKING NONACCIDENTAL INJURY

Traumatic and nontraumatic conditions may mimic the clinical presentations (ie, the triad) and imaging findings of NAI. These include accidental trauma (as previously discussed), birth trauma, hypoxia-ischemia, cardiopulmonary

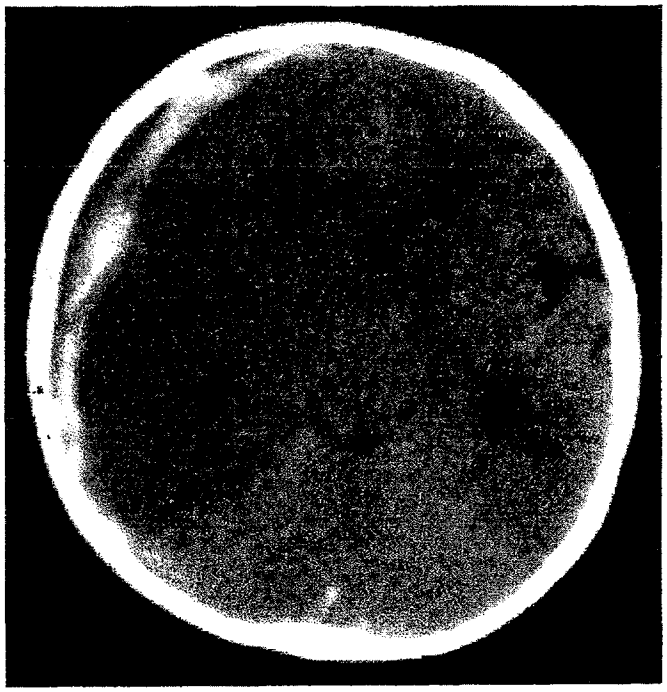

FIGURE 17. Image obtained from an 8-month-old male infant who had ALTE, right-side occipital skull fracture (not shown), a healing right-side distal radial fracture, and then had brain death. Computed tomographic image shows a right-side, mixed-density extracerebral collection with right-side cerebral low density, mass effect, and leftward shift. High-density hemorrhages (or thromboses) are also present along the tentorium. There was disagreement among the forensic experts regarding hyperacute-acute SDH versus chronic SDH with rehemorrhage.

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resuscitation, infectious or postinfectious conditions (eg, sepsis, meningoencephalitis, postvaccinial), vascular diseases, coagulopathies, venous thrombosis, metabolic disorders, neoplastic processes, certain therapies, extracorporeal membrane oxygenation (ECMO), and other conditions.[4,5,10,114,115,133] Regarding the pathogenesis of the triad (with and without other organ system involvement [eg, skeletal]), and whether caused by NAI, AI, or nontraumatic etiologies, the pathophysiology seems to be some combination or sequence of factors, including increased intracranial pressure, increased venous pressure, systemic hypotension or hypertension, vascular fragility, hematologic derangement, and/or collagenopathy superimposed on the immature CNS and other systems.[107,115,123,132–146]

Although the initial medical evaluation, including history, laboratory tests, and imaging studies, may suggest an alternative condition, the diagnosis may not be made because of a *rush to judgment* regarding NAI. It is important to be aware of these mimics because a more extensive workup may be needed beyond the routine *screening* tests. In addition, the lack of confirmation of a specific condition does not automatically indicate the *default* diagnosis of NAI. In all cases, it is critical to review all records dating back to the pregnancy and birth, the postnatal pediatric records, the family history, the more recent history preceding the short-term presentation, the details of the short-term event itself, the resuscitation, and the subsequent management, all of which may contribute to the clinical and imaging findings.[4,5,10,115,133]

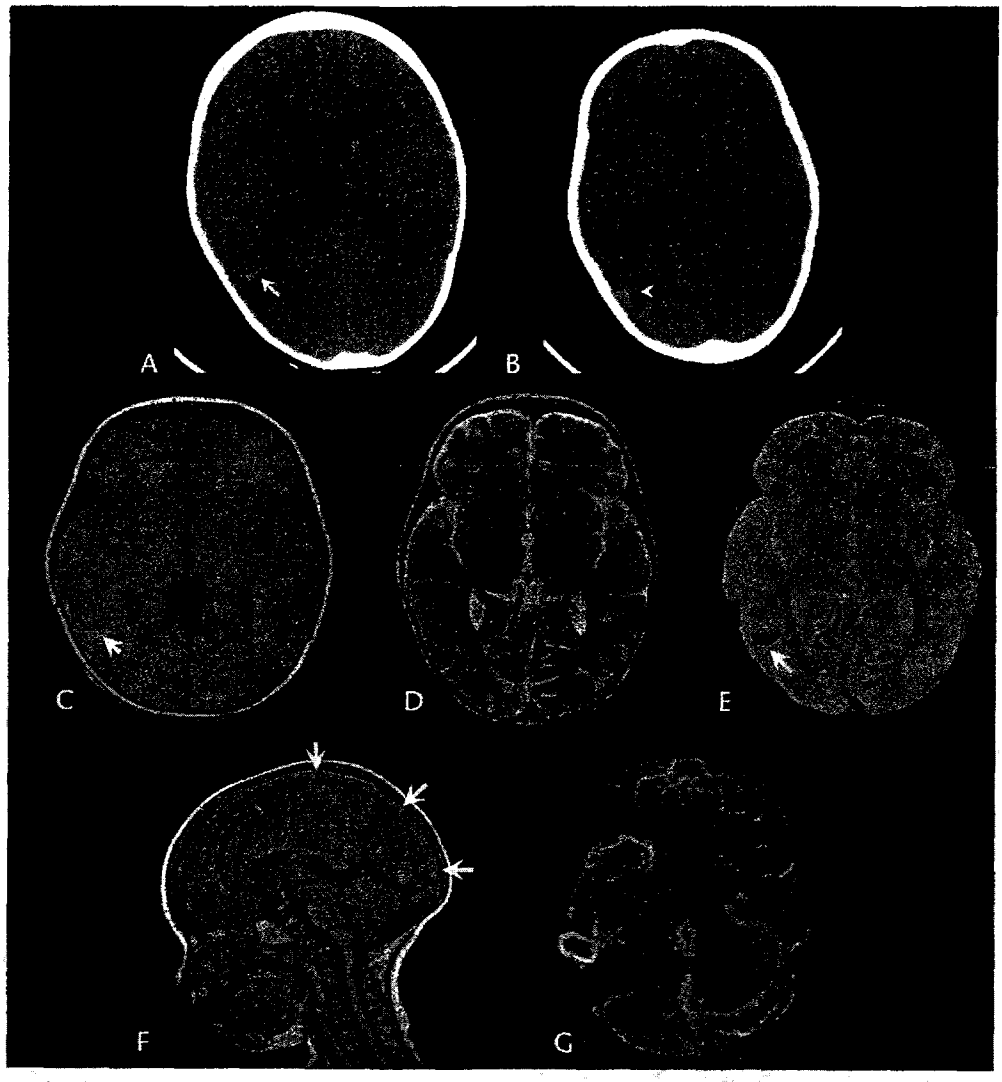

FIGURE 18. Images obtained from a 22-month-old boy who experienced lethargy, vomiting, and seizures after a viral illness, plus thrombocytopenia and iron deficiency anemia. A–B, Computed tomographic images show right-side posterior temporal and peritentorial high-density foci of hemorrhage or thrombosis (arrows). Axial T1 (C), T2 (D), and GRE (E) images show corresponding T1-hyperintense and GRE-hypointense foci with associated T2 hyperintensity (arrows). F, Sagittal T1 MRI scan shows hyperintensity along the superior sagittal sinus (arrows [thrombosis vs slow flow]). G, Axial MRV projection image shows nonvisualization of the superior sagittal, right-side transverse, and right-side sigmoid sinuses (diagnosis, postviral dural and cerebral venous thrombosis [extensive coagulopathy workup continues]).

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A recent review presented by Sirotnak[133] extensively catalogues the many conditions that may mimic abusive head trauma. These include perinatal conditions (birth trauma and congenital malformations), accidental trauma, genetic and metabolic disorders, hematologic diseases and coagulopathies, infectious diseases, autoimmune and vasculitic conditions, oncological disease, toxins, poisons, nutritional deficiencies, and medical and surgical complications. The reader is encouraged to read this review.[133] An abbreviated discussion is presented in this article along with some examples.

Birth Trauma and Neonatal Conditions

Manifestations of birth trauma, including Fx, SDH, and RH, may persist beyond the neonatal period and mimic CNS findings of abuse.[145–151] Other examples are the cases of infants following ECMO therapy, at-risk preterm neonates, and infants with congenital heart disease.[4,5,10,123,124,152] When evaluating the condition of a young infant with apparent NAI, it is important to consider that the clinical and imaging findings may actually stem from parturitional and neonatal issues. This includes hemorrhage or rehemorrhage into collections existing at birth (Figs. 5, 8, 13).

Developmental Anomalies

Vascular malformations of the CNS in neonates and infants are relatively rare.[115,133,153,154] The most common are the vein of Galen malformations. Aneurysms are also rare in childhood but may arise within the circle of Willis. Aneurysms outside the circle are usually mycotic or traumatic in origin. Increased risk of aneurysm is associated with certain conditions, such as coarctation of the aorta, polycystic kidney disease, neurofibromatosis, and a family history positive for aneurysm. A number of syndromes in childhood are associated with vascular anomalies and may present with intracranial hemorrhage. These syndromes include, as examples, PHACE (*p*osterior fossa brain malformations, *h*emangiomas, *a*rterial anomalies, *c*oarctation of the aorta, *c*ardiac defects, and *e*ye abnormalities), Sturge-Weber, Beckwith-Wiedemann, Klippel-Trenaunay-Weber, Maffucci, and Olser-Weber-Rendu. Arachnoid cysts are also known to be associated with SDH and RH, spontaneously and with trauma (Fig. 19).[133,155]

Genetic and Metabolic Disorders

A number of conditions in this category may present with intracranial hemorrhage (eg, SDH) or RH. These include osteogenesis imperfecta, glutaric aciduria type I, Menkes kinky hair disease, Ehlers-Danlos and Marfan syndromes, homocystinuria, and others (Fig. 19).[115,133,135,136,156]

Hematologic Disease and Coagulopathy

Many conditions in this category predispose to intracranial hemorrhage and RH.[4,5,10,114,115,133,140–143,157] The bleeding or clotting disorder may be primary or

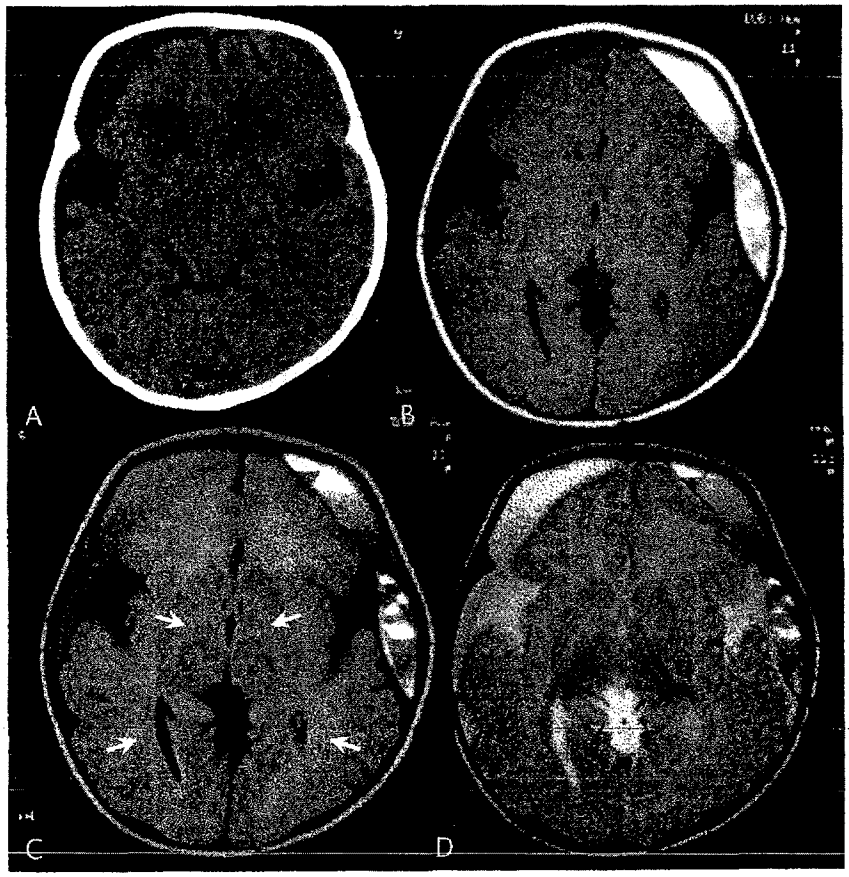

FIGURE 19. Images obtained from a 9-month-old male infant with glutaric aciduria type 1, SDHs, and RHs. CT (A), T1 (B), FLAIR (C), and T2 (D) MRI images show bilateral mixed-density and mixed-intensity extracerebral collections with fluid levels and septations, especially on the left side. Other characteristic findings for glutaric aciduria type 1 include bilaterally wide sylvian fissures (arachnoid cysts) plus abnormal basal ganglia (globus pallidus) and cerebral white matter intensities (arrows).

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FIGURE 20. Images obtained from a 1-week-old male neonate with seizures, thrombocytopenia, antithrombin III deficiency, and ECMO for pulmonary hypertension. Axial T2 FSE (A) and GRE (B) MRI images show bilateral, mixed-intensity SDHs (arrows).

secondary (Figs. 7, 14–16, 18, 20, 21). In some cases, a more extensive workup beyond the usual *screening* tests will be needed, including a hematology consultation. Included in this category are the anemias, hemoglobinopathies (eg, sickle cell disease), hemorrhagic disease of the newborn (vitamin K deficiency Fig. 21), hemophilia A and B, factor V and XII deficiencies, von Willebrand disease, idiopathic thrombocytopenic purpura, disseminated intravascular coagulation and consumption coagulopathy associated with other conditions (eg, trauma, infection), liver disease, nephrotic syndrome, hemophagocytic lymphohistiocytosis, anticoagulant therapy, and others. Venous thrombosis may involve the dural venous sinuses (ie, dural venous sinus thrombosis [DVST]) and/or the cerebral veins (ie, cerebral vein thrombosis [CVT]) and be associated with primary or secondary hematologic or coagulopathic state.[10,123,124,133,158–161] Risk factors include acute systemic illness, dehydration (fluid-electrolyte imbalance), sepsis, perinatal complications, chronic systemic disease, cardiac disease, connective tissue disorder, hematologic disorder, oncological disease and therapy, head and neck infection, and hypercoagulable states. Seizure and/or neurological deficit are common, and hemorrhagic infarction is characteristic. Subarachnoid hemorrhage, SDH, or RH may also be observed, especially in infants (Figs. 15, 16, 18, 22). Relative high densities anywhere along the dural venous sinuses, tentorium, and falx (interhemispheric fissure and inferior sagittal sinus) may be seen on initial CT. Linear high densities may also be present along the distribution of the cortical ("cord sign"), subependymal, or medullary veins and give the impression of SAH, SDH, or intracerebral

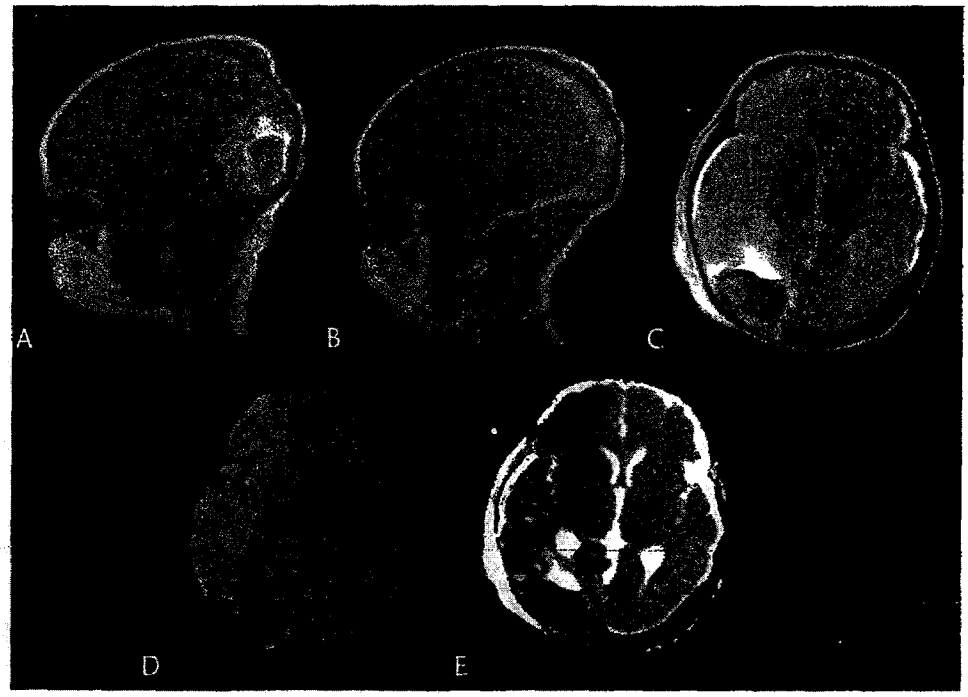

FIGURE 21. Images obtained from a 1-week-old male neonate who had seizures after delivery at home (no vitamin K administered). After surgical evacuation of large, right-side SDH, sagittal T1 (A, B), axial T2 (C), ADC (D), and DWI (E) images show bilateral mixed-intensity extracerebral and intracerebral hemorrhages and right-side cerebral hemispheric restricted diffusion (likely infarction) (diagnosis, hemorrhagic disease of the newborn [vitamin K deficiency]).

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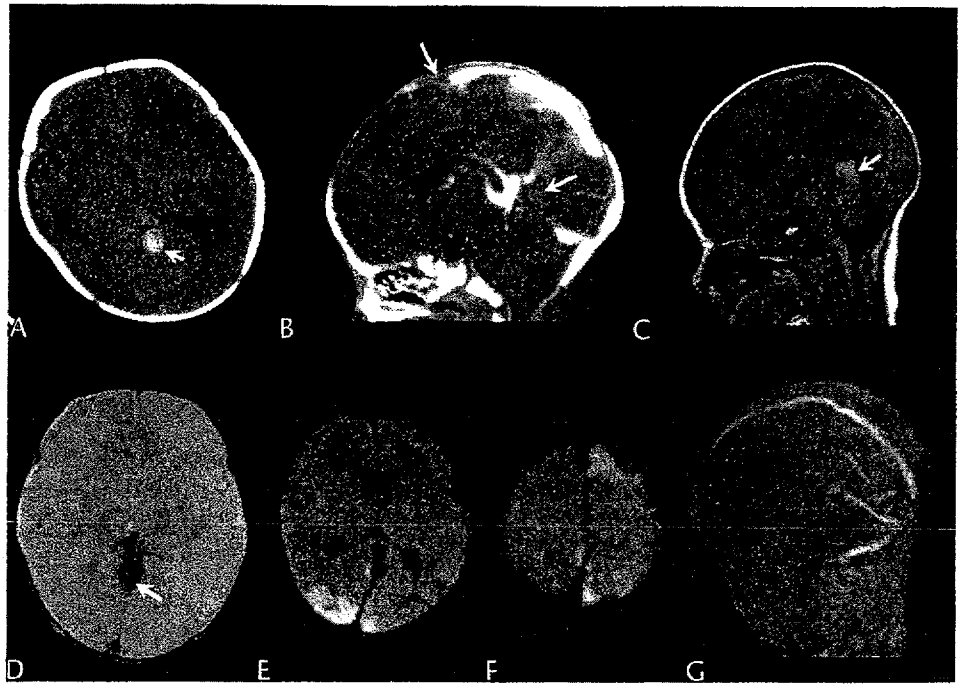

FIGURE 22. Images obtained from a 2-week-old male neonate with lethargy in emergency room (ER). Computed tomographic image (A) shows a focal midline hyperdensity at the level of the straight sinus (arrowhead). Sagittal CTV image (B) shows luminal masses along the straight and superior sagittal sinuses (arrows). Sagittal T1 (C) and axial GRE (D) images show the thrombus within the straight sinus (arrows). Axial DWI images (E–F) show restricted diffusion in multiple cortical areas (likely infarction vs suppuration). Magnetic resonance venography (G) is of poor diagnostic quality as compared with CTV (diagnosis, group B streptococcal meningitis with DVST).

hemorrhage. The "empty delta" sign may be seen within the superior sagittal sinus on contrast-enhanced CT. There may be multifocal infarctions (hemorrhagic or nonhemorrhagic) or intraventricular hemorrhage. With extensive dural venous sinus or cerebral venous thrombosis, there may be massive, focal, or diffuse edema. Orbit, paranasal sinus, or otomastoid disease may be associated with basal venous sinus thrombosis (eg, cavernous, petrosal, sphenoparietal). The thromboses and associated hemorrhages have variable MRI appearance depending on their age (see Image Analysis–Magnetic Resonance Imaging section and Table 1). Computed tomographic venography or MRV may readily detect DVST but not cerebral vein thrombosis, which may be suspected because of the characteristic distribution of hemorrhage or thromboses along venous structures, as demonstrated on susceptibility-weighted sequences (eg, GRE hypointensity). Depending on the clinical context, treatment may be directed only to the specific cause (eg, infection) or may also include anticoagulation or thrombolysis.

Infectious and Postinfectious Conditions

Meningitis, encephalitis, or sepsis (eg, bacterial, viral, granulomatous, parasitic) may involve vascular structures resulting in vasculitis, arterial or venous thrombosis, mycotic aneurysm, infarction, and hemorrhage (Figs. 3, 14–17, 22, 23). Subdural hemorrhage and RH may also be observed.

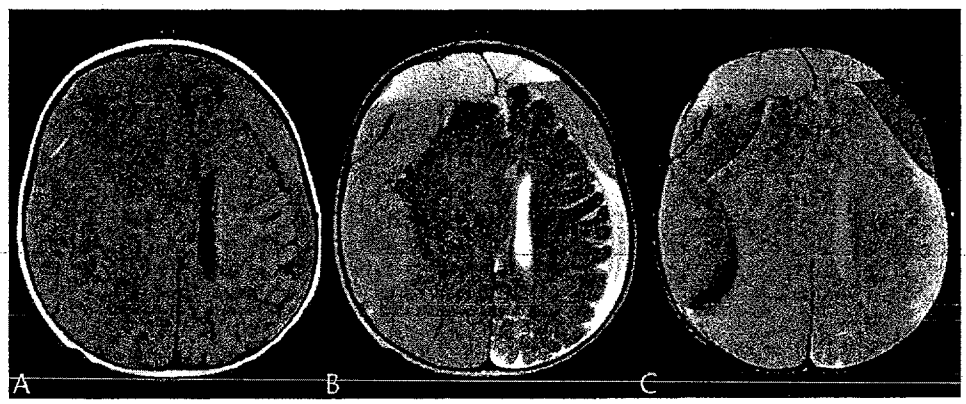

FIGURE 23. Images obtained from a 5-month-old male infant who had macrocephaly and seizures after having group D streptococcal (nonenterococcal) meningitis at the age of 3 days. Axial T1 (A), T2 (B), and GRE (C) images show bilaterally large and mixed-intensity extracerebral collections with septations and asymmetrical mass effect (likely chronic subdural effusions or hygromas with rehemorrhage).

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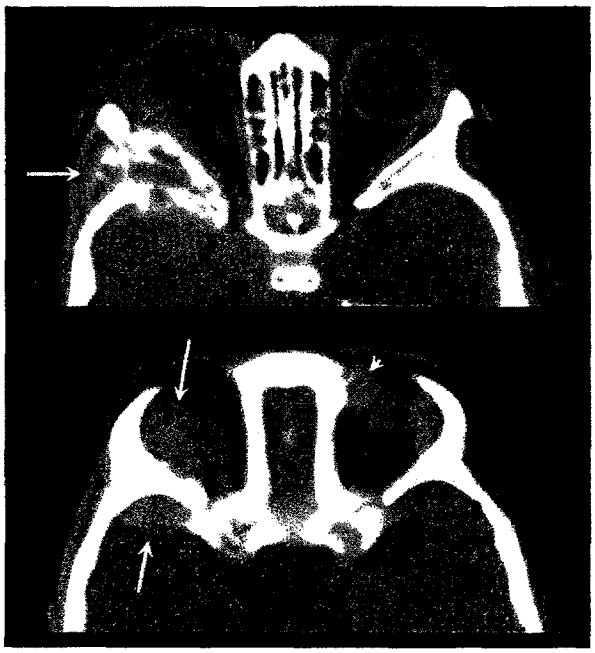

FIGURE 24. Images obtained from an 18-month-old girl with periorbital and facial ecchymoses in ER, evaluated for NAI. Computed tomographic image shows bilateral iso-high-density orbital soft tissue masses with bone destruction (arrows) and extension into the right-side middle cranial fossa (diagnosis, neuroblastoma).

Postinfectious illnesses (eg, postvaccinial) may also be associated with these findings.[139] Included in this category are the *encephalopathies of infancy and childhood* and *hemorrhagic shock and encephalopathy syndrome*.[115,133]

Autoimmune and Vasculitic Conditions

These include Kawasaki disease, systemic lupus erythematosis, moyamoya disease, Wegener granulomatosis, and Behçet syndrome.[115,133]

Oncological Disease

Hematologic malignancies, solid tumors of childhood, and their attendant therapies (including transplantation) are commonly associated with a variety of sequelae or complications that predispose to hemorrhage (eg, SDH and RH).[115,133] This includes vascular invasion by tumor, immunocompromise, infection, and coagulopathy. The clinical presentation and image findings may be mistaken for NAI (eg, leukemia, neuroblastoma) (Fig. 24).

Toxins, Poisons, and Nutritional Deficiencies

This category includes lead poisoning, cocaine, anticoagulants, and vitamin deficiencies (eg, vitamins K, C, D) (Figs. 21, 25). Preterm neonates and other chronically ill infants are particularly vulnerable to nutritional deficiencies and complications of prolonged immobilization that often primarily affect bone development. Such infants may have skeletal imaging findings (eg, multiple healing fractures) that are misinterpreted as NAI, particularly if they present with AI that is complicated by SDH and RH (Fig. 25).[162–174]

Medical and Surgical Complications

This category includes (1) anticoagulant therapy or treatment-induced coagulopathy and (2) morbidity from medical or surgical interventions.[115,133]

CONCLUSIONS

In view of the currently available data, it is clear that we do not have an established EBM platform from which to

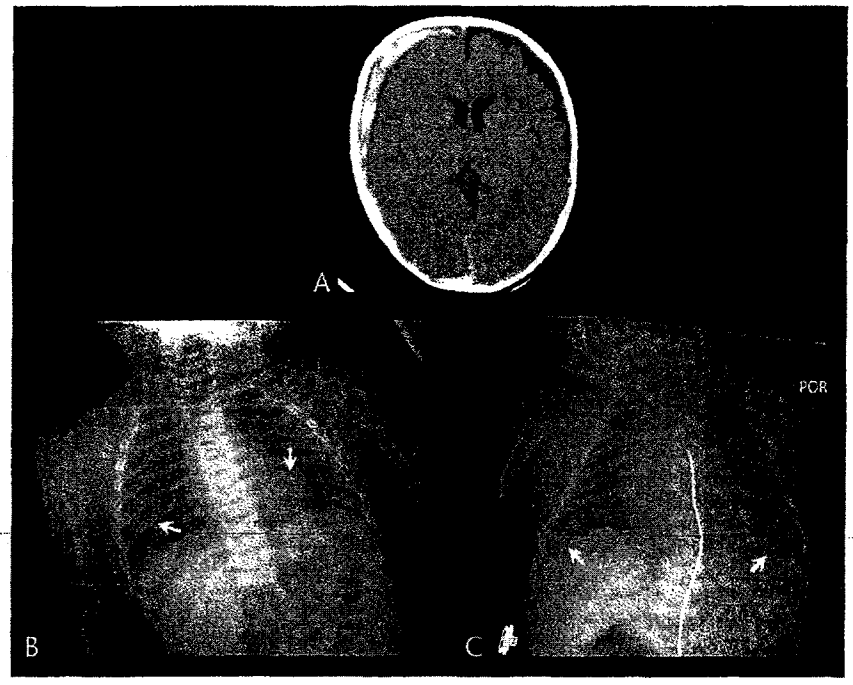

FIGURE 25. Images obtained from a 7-month-old male infant (25-week preterm birth) dropped with head impact to floor, RHs, evaluated in ER. Computed tomographic image (A) shows right-side mixed high-density extracerebral collection, left-side low-density extracerebral collection, posterior interhemispheric high-density hemorrhage, and right-side cerebral low-density edema. Chest radiograph in ER (B) shows bilateral anterior and posterior old, healing rib fractures. Comparison with earlier chest radiograph (C) at discharge from neonatal intensive care unit shows diffuse osteopenia and anterior rib flaring (arrows). Diagnosis: rickets of prematurity vs NAI?; AI with acute SDH superimposed on BECC vs NAI?

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distinguish NAI from AI and, in some cases, traumatic from nontraumatic CNS injury. More reliable research is needed to establish a sound scientific foundation for CNS injury in NAI. The young infant is assumed more vulnerable to traumatic CNS injury, whether accidental or not, as compared with the older child or adult, and relies on the attention of caretakers for safety. However, as the infant becomes more mobile (rolling, crawling, walking, etc), the risk of AI (eg, from falls) increases. Furthermore, the interaction with older siblings or other children becomes a factor. The medical and imaging findings cannot diagnose intentional injury. Only the child protection investigation may provide the basis for inflicted injury in the context of supportive medical, imaging, or pathological findings. Furthermore, biomechanical factors must be taken into consideration regarding the mechanism of trauma.

The radiologist should describe the imaging findings in detail, including the pattern, distribution, and severity of injury. A DDX is given, and timing ranges are provided if possible. If NAI is at issue, then the radiologist must directly communicate the imaging findings to the primary care team and be available to consult with child protection services and other medical or surgical consultants, including the pathologist or biomechanical specialist, law enforcement investigators, and attorneys for all parties, as appropriate.[1–5] The pattern of injury and the timing parameters, as may be provided by MRI, are particularly important with regard to correlation of events as reported by witnesses and potential suspects. The radiologist must also be aware of certain conditions that are known to have clinical and imaging features that may mimic abuse.[1–5] These should be properly ruled out, and the possibility of combined or multifactorial mechanisms with synergistic effects should also be considered (eg, predisposing condition plus trauma). A timely and thorough multidisciplinary evaluation may be the difference between an appropriate child protection and an improper breakup of the family or a wrongful indictment and conviction.

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Ex. 2
Second Affidavit of Dr. Patrick Barnes.